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Acute and episodic vestibular syndromes caused by ischemic stroke: predilection sites and risk factors

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Abstract

Objective: This study was performed to investigate the predilection sites of acute vestibular syndrome (AVS) and episodic vestibular syndrome (EVS) caused by acute infarcts.

Methods: This retrospective cohort study was performed at a stroke center in a tertiary teaching hospital. We diagnosed patients with AVS/EVS caused by acute ischemic stroke using diffusion-weighted imaging (DWI) and magnetic resonance angiography.

Results: Among all patients with AVS/EVS, 68 had DWI-positive ischemic events and 113 had DWI-negative ischemic events. Of the 68 patients with positive DWI findings, 42.6% had acute infarcts in the anterior circulation and 41.2% had acute infarcts in the posterior circulation. The main stroke predilection sites were the insular cortex (22.1%) and posterior thalamus (11.8%). Large vessel stenosis/occlusion (odds ratio, 0.12; 95% confidence interval, 0.04–0.36) and focal neurological symptoms/signs (odds ratio, 0.27; 95% confidence interval, 0.10–0.72) were significantly associated with the risk of AVS/EVS in patients with acute ischemic stroke.

Conclusions: The main predilection sites of AVS/EVS caused by ischemic stroke are the insular cortex and posterior thalamus. The risk of AVS/EVS is associated with large vessel stenosis and focal symptoms.

Keywords

Acute ischemic stroke, acute vestibular syndrome, episodic vestibular syndrome, diffusion-weighted imaging, magnetic resonance angiography, insular cortex, posterior thalamus

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Introduction

Although acute vestibular syndrome (AVS) has been described in previous reports,[1,2] a new approach in the 11th edition of the International Classification of Diseases (ICD-11) divides patients with vertigo or dizziness into two key categories: those with AVS, which is defined as a clinical syndrome of acute-onset, continuous vertigo, dizziness, or unsteadiness lasting days to weeks and generally including features suggestive of new, ongoing vestibular system dysfunction (e.g., vomiting, nystagmus, or severe postural instability), and those with episodic vestibular syndrome (EVS), which is defined as a clinical syndrome of transient vertigo, dizziness, or unsteadiness lasting seconds to hours (occasionally days) and generally including features suggestive of temporary, short-lived vestibular system dysfunction (e.g., nausea, nystagmus, or sudden falls). Anatomically, AVS and/or EVS may occur at any point along the vestibular pathway from the peripheral labyrinth to the central vestibular cortex. Therefore, the causes of vestibular syndrome are divided into two categories: peripheral causes (e.g., vestibular neuronitis, acute labyrinthitis, benign paroxysmal positional vertigo [BPPV], and Ménière's disease) and central causes (e.g., traumatic vestibulopathy, demyelinating disease with vestibular involvement, and stroke affecting central vestibular structures). Central vestibular syndrome is defined as AVS and/or EVS with a central cause.[1,3] Previous studies have suggested that isolated vertigo or dizziness is a symptom of posterior circulation disruption.[4,5] However, other studies have shown that such symptoms may indicate involvement of the anterior circulation.[6,7] The richest vestibular pathways in humans are the cortical and subcortical areas of both hemispheres.[8] However, few reports have provided detailed information about the predilection sites of central AVS and/or EVS. We hypothesized that evidence of ischemic events on diffusion-weighted imaging (DWI) in patients with AVS and/or EVS accurately represents the predisposing site of acute vestibular damage. Therefore, this study was performed to determine whether magnetic resonance imaging (MRI)-DWI combined with magnetic resonance angiography (MRA) can clarify the predilection sites of acute ischemic stroke causing AVS and/or EVS.

Methods

Study participants

This retrospective observational study involved consecutive adult patients with acute vertigo or dizziness that met the diagnostic criteria for AVS and/or EVS who underwent MRI in the neurologic ward and stroke center of Shuyang County Hospital in Northern China from March 2014 to March 2016. The study was approved by the Ethics Committee on Clinical Research of Shuyang Hospital, Xuzhou Medical University. Because of the retrospective nature of this study, the requirement for informed consent was waived.

This study involved patients with AVS and/or EVS, defined as follows: (1) acute onset of dizziness/vertigo consistent with the standard definition of AVS and/or EVS published by the ICD-11,[9] (2) confirmation of an acute ischemic stroke event by DWI or observation of a recent ischemic lesion by fluid-attenuated inversion recovery (FLAIR) imaging within 24 to 72 hours after symptom onset, and (3) no history of recurrent dizziness or vertigo. The exclusion criteria were peripheral vestibular syndrome (e.g., BPPV or Ménière's

disease),[10] migrainous vertigo, spontaneous intracerebral hemorrhage, vertebrarterial type cervical spondylopathy, demyelinating disease, drug-induced causes, and other causes of non-ischemic stroke.

The study population was divided into two groups according to confirmation of an increased signal on DWI consistent with a diagnosis of an acute infarct: the DWI-positive group and the DWI-negative group.

MRI protocol

In all patients, MRI was performed with a 1.5-T MRI machine (Siemens AG, Munich, Germany). MRI sequences included conventional T2-weighted imaging, T1-weighted imaging, FLAIR imaging, and DWI in the axial view from medulla to cortex with a 5-mm section thickness. MRA was used for three-dimensional time-of-flight imaging on an axial scan with a −0.6-mm interval and slice thickness of 1.1 mm. The range of brain MRA included the main branches and trunk in the anterior and posterior circulation as well as the entire circle of Willis.

Patient assessment

We referred to the Oxfordshire Community Stroke Project classification system of stroke. The diagnosis of ischemic infarction in a vascular region of the brain was divided into two regions: anterior circulation artery (ACA) and posterior circulation artery (PCA). Symptomatic carotid artery or vertebral basilar artery stenosis was defined as a $\geq$50% reduction in the diameter of the carotid artery or vertebral basilar artery at a location considered likely to be responsible for the patient's acute infarct or clinical symptoms. Based on the classification of subtypes in acute ischemic stroke established by the Trial of ORG 10172 in Acute Stroke Treatment,[11] a large infarct was defined as a >15-mm-diameter lesion on brain MRI-DWI and a small infarct (lacunar infarct) was defined as a $\leq$15-mm-diameter lesion.

Clinically, isolated AVS and/or EVS is caused by a pure vestibular structure lesion, while non-isolated AVS and/or EVS is caused by a vestibular structure lesion with focal symptoms or signs due to another type of neurological structure lesion. We defined a transient ischemic attack (TIA) as a <24-hour episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia without a recent or new infarction.

Data collection

The initial MRI-based diagnosis was determined by a highly qualified radiologist. The MRI-DWI infarct diameter (mm) in each MRI slice was measured by a highly qualified neurologist and was assessed in combination with each patient's detailed medical records and lesion position. The MRA images were assessed and the location and abnormal condition of blood vessels were registered by the same highly qualified neurologist. We also analyzed each patient's age, history of hypertension, history of diabetes mellitus, atrial fibrillation or history of heart disease, drinking habits, and smoking habits. Moreover, we collected the neurological examination findings, including focal neurological symptoms and signs, bedside oculomotor examination results (i.e., head impulse test, nystagmus assessment, and skew deviation),[10] and laboratory examination findings. For the outcome analyses, a neurologic specialist assessed the modified Rankin scale score at the 30-day follow-up. This score ranges from 0 to 6 (0, no symptoms; 1, slight symptoms; 2, restriction; 3, slight disability; 4 moderate disability; 5, severe disability; and 6, death).

Statistical analysis

The measurement data of all patients in this study are expressed as mean ± standard deviation, median (range), or number (percentage), and the count data were measured by a *t* test. Multivariable logistic regression was used to determine the risk factors for stroke causing AVS and/or EVS. The data were analyzed using SPSS v.17.0 (SPSS Inc., Chicago, IL, USA). A p value of <0.05 was considered statistically significant.

Results

In total, 502 adult patients with acute vertigo or dizziness who met the diagnostic criteria for AVS and/or EVS and underwent MRI were evaluated. After excluding 186 patients with peripheral causes such as BPPV, 71 with migrainous vertigo, 31 with intracerebral hemorrhage, 14 with vertebrarterial type cervical spondylopathy, 10 with demyelinating disease, 6 with drug-induced causes, and 3 with other causes of non-ischemic stroke, 181 patients who met the inclusion criteria were enrolled in the present study. Of these 181 patients with AVS and/or EVS caused by ischemic events, 68 had DWI-positive ischemic events (acute infarcts) and 113 had DWI-negative events (e.g., ischemic events on FLAIR, TIA, septic shock, or cardiac arrhythmia) as observed by MRI (98.5%) and MRA (90.3%).

Among the 68 (37.6%) patients with DWI-positive ischemic events causing central AVS and/or EVS, the median time from emergency room presentation to hospital admission was 1.5 days (range, 30 minutes to 14 days). The median time from symptom onset to MRI-DWI was 1.5 days (range, 0.2–14.8 days). The imaging changes and clinical features of the 68 patients with AVS and/or EVS caused by acute ischemic stroke are shown in Table 1.

Table 1. Clinical findings and DWI characteristics of 68 patients with AVS/EVS caused by acute infarction.

Characteristics	Value
Sex	
Female	23 (33.8)
Male	45 (66.2)
Age, years	63.5 (32–85)
Time from symptom onset to MRI, days	1.5 (0.2–14.8)
Rapid-onset vestibular symptoms	
Isolated AVS/EVS	46 (67.6)
AVS	15 (32.6)
EVS	31 (67.4)
Non-isolated AVS/EVS	22 (32.4)
AVS	12 (54.5)
EVS	10 (45.5)
Head motion intolerance	49 (72.0)
Gait unsteadiness	48 (70.6)
Nausea or vomiting	47 (69.1)
Nystagmus	
Spontaneous nystagmus	9 (13.2)
Positive head impulse test	13 (19.1)
Positive ocular tilt reaction	6 (8.8)
Focal neurological symptoms/signs	
Hemiparesis	8 (11.8)
Numbness of limbs	5 (7.4)
Slurred speech	5 (7.4)
Hemianopsia	2 (2.9)
Other	2 (2.9)
MRI-DWI	
Size of lesion, mm	4 (0.6–89.4)
Number of lesions	2 (1–26)
Small infarcts	58 (85.3)
ACA infarcts	29 (42.6)
PCA infarcts	28 (41.2)
ACA + PCA infarcts	11 (16.2)
Main predilection sites of stroke	
Insular cortex	15 (22.1)
Posterior thalamus	8 (11.8)
Large artery stenosis on MRA	26 (38.2)
ACA	12 (46.2)
PCA	9 (34.6)
ACA + PCA	5 (19.2)
Disabling	15 (22.1)
Mortality in 30 days	1 (1.5)

Data are presented as n (%) or median (range). AVS, acute vestibular syndrome; EVS, episodic vestibular syndrome; ACA, anterior circulation artery; PCA, posterior circulation artery; MRI, magnetic resonance imaging; MRA, magnetic resonance angiography.

Pure ACA region infarcts were present in 42.6% (29/68) of the patients. The most frequent location was the insular cortex (22.1%, 15/68). Of these patients, 14 had a

unilateral insular lesion, and the majority of lesions were located in the posterior region of the insular cortex (Figure 1). Non-isolated insular infarcts were found in more than half of these patients (8/15).

Other types of cerebral lobe infarcts in the ACA were also common (Figure 2), including infarcts in the frontal lobe in four patients; parietal lobe in three; frontal, temporal, and parietal lobes in two; temporal and parietal lobes in two; temporal lobe in one; and periventricular region in two. An ipsilateral large vessel stenosis/occlusion in the ACA as confirmed by MRI was

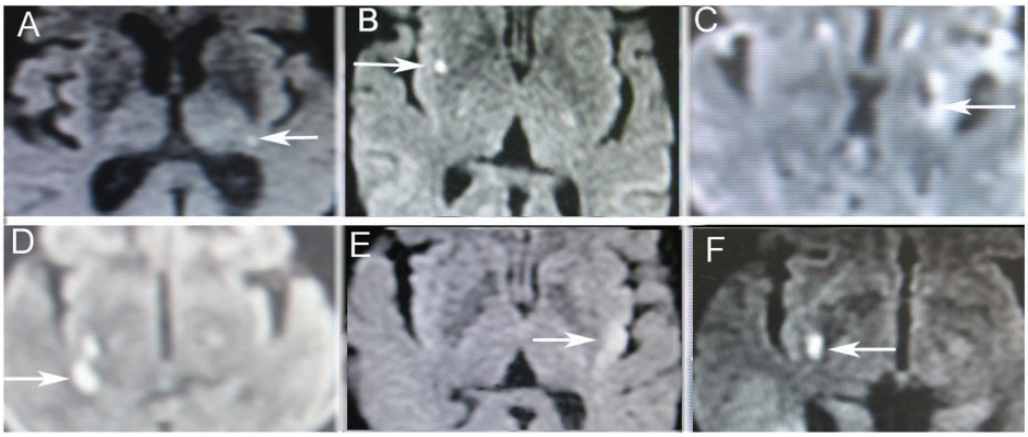

Figure 1. Magnetic resonance imaging–diffusion-weighted imaging findings of acute insular infarcts in 15 of 68 patients with central acute vestibular syndrome/episodic vestibular syndrome. Only six select images representing different involved topographical regions are shown. (a, b, c, f) The median-posterior insular infarcts (arrows) are small and elliptical. (d, e) Dorsal insular infarct (arrow).

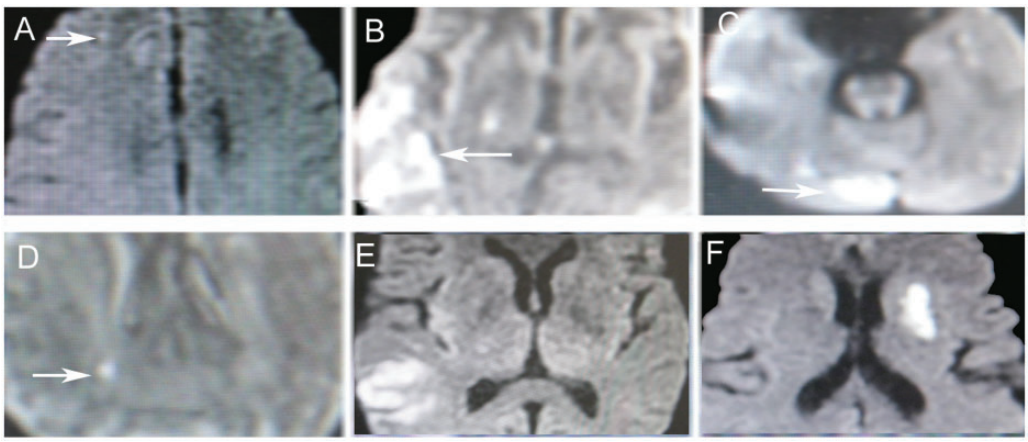

Figure 2. Magnetic resonance imaging–diffusion-weighted imaging findings of acute cerebral lobe infarcts in 14 of 68 patients with central acute vestibular syndrome/episodic vestibular syndrome. Only six select images representing different involved topographical regions are shown. (a) Frontal lobe infarct (arrow). (b) Temporal parietal lobe infarct (arrow). (c, d) Occipital lobe infarct (arrow). (e) Parietal lobe infarct (arrow). (f) Left frontal lobe infarct (arrow) spreading to regions of the insular lobe.

present in 12 (46.2%) patients, including an ipsilateral middle cerebral artery stenosis/occlusion in 6, ipsilateral internal carotid artery occlusion in 4, and ipsilateral anterior cerebral artery occlusion in 2.

Pure posterior circulation infarcts were present in 41.2% (28/68) of the patients. DWI showed that in eight patients, the impaired thalamic vestibular structure was most commonly limited to the posterolateral thalamus (Figure 3). All eight patients had unilateral lesions; of these, an isolated lesion was present in only two patients.

The other cerebral infarcts in the PCA were present in the pons in seven patients, cerebellum in five, occipital lobe in four, midbrain in three, and medulla in one. Among the patients with posterior circulation infarcts, nine (34.6%) had ipsilateral vertebral basilar artery stenosis/occlusion as confirmed by MRA.

The ACA and PCA were involved in 11 (16.2%) patients, including the parietal lobe and cerebellum in 3; temporal, parietal, and occipital lobes in 2; parietal lobe and medulla in 2; cingulum in 2; and medulla plus cerebellum in 2. Among these patients, five had large vascular stenosis/occlusion, including the unilateral middle cerebral artery in three, unilateral superior cerebellar artery in one, and unilateral vertebral artery in one.

DWI showed that the median infarct diameter was 4.0 mm (range, 0.6–89 mm). Small infarcts confirmed by DWI were present in 58 (85.3%) patients, and only 18 (31.0%) patients had ipsilateral large vessel stenosis or occlusion as confirmed by vascular imaging (Figure 4). The infarct lesions were >15 mm in 10 patients, among whom MRA showed ipsilateral large vessel stenosis or occlusion in 8 patients. The details of the other two patients were unknown.

The univariate analysis revealed a significantly increased risk of ischemic events in patients with DWI-positive findings and the following risk factors: age of >60 years (60.3% vs. 44.2%, $p = 0.048$), high systolic blood pressure (151.2 ± 22.5 vs. 143.8 ± 16.0 mmHg, $p = 0.011$), focal neurological symptoms or signs (32.4% vs. 8.8%, $p = 0.000$), and large vessel stenosis (38.2% vs. 5.3%, $p = 0.000$) (Table 2). However, the other risk factors were not significantly different

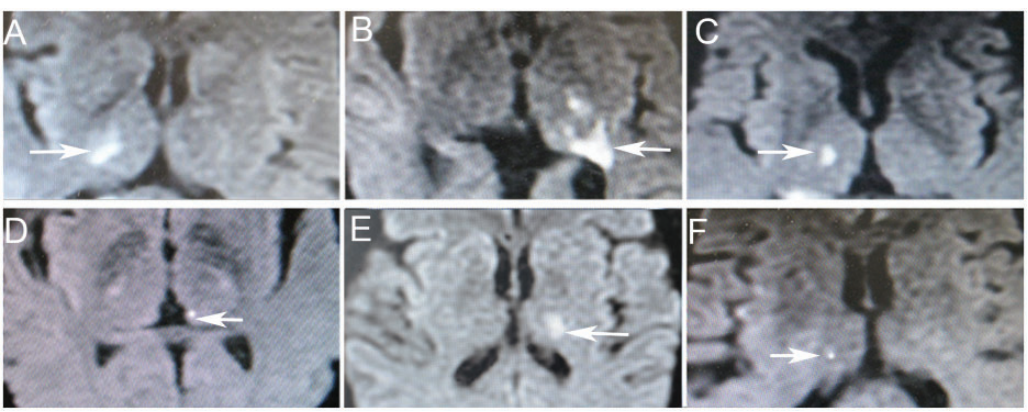

Figure 3. Magnetic resonance imaging–diffusion-weighted imaging showing acute thalamic vestibular infarcts in 8 of 68 patients with central acute vestibular syndrome/episodic vestibular syndrome. Six select images representing different involved topographical regions are shown. (a, b, c, e, f) Thalamic vestibular structure infarcts were usually limited to the posterolateral thalamus (arrows). (d) Only one patient had a vestibular lesion limited to the dorsal region of the thalamus (arrow).

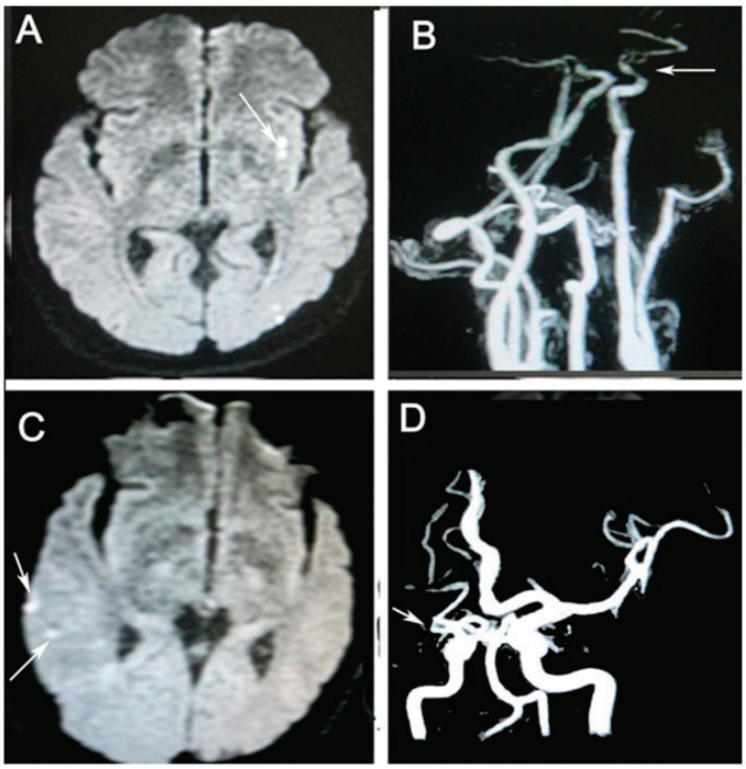

Figure 4. Diffusion-weighted imaging (DWI) and vascular imaging of acute infarction in the vestibular throwing structure of the anterior circulation. A 40-year-old man presented with a 4-day history of acute vestibular syndrome and slightly slurred speech; (a) head magnetic resonance imaging (MRI)-DWI showed an acute small infarct in the left insula (arrow), and (b) computed tomography vascular imaging showed the beginning of the left internal carotid artery stenosis (arrows). A 55-year-old man presented with a 1-day history of episodic vestibular syndrome; (c) MRI-DWI showed an acute small infarction (arrow) in the right temporal lobe, and (d) magnetic resonance angiography showed an occlusion in the right side of the middle cerebral artery (arrow).

between the groups. The results of the multivariate regression analysis showed that only large vessel stenosis or occlusion (odds ratio, 0.12; 95% confidence interval, 0.04–0.36) and focal neurological symptoms or signs (odds ratio, 0.27; 95% confidence interval, 0.14–0.72) were independent risk factors for AVS and/or EVS in patients with evidence of acute ischemic events on DWI (Table 3).

In the outcome analyses, 15 of the 68 patients with AVS and/or EVS who had DWI-positive ischemic events had slight to moderate disability (modified Rankin scale score of 3–4) at the 30-day follow-up after the initial event compared with 8 of the 113 patients with AVS and/or EVS who had DWI-negative ischemic events (22.1% vs. 7.1%, $p = 0.001$). Only one patient in the DWI-positive group had died by the 30-day follow-up.

Discussion

In the present brain DWI study, the cause of AVS and/or EVS was an acute ischemic

Table 2. Comparison of risk factors and associated events between patients with AVS/EVS showing positive and negative DWI signs.

Episodes	AVS/EVS with positive DWI signs (n = 68)	AVS/EVS with negative DWI signs (n = 113)	p value
Traditional risk factors			
Male sex	45 (66.2)	86 (76.1)	0.171
Age, years	62.9 ± 11.3	61.3 ± 12.0	0.580
Age of >60 years	41 (60.3)	50 (44.2)	0.046
History of hypertension	50 (73.5)	67 (59.3)	0.056
Diabetes mellitus	20 (29.4)	31 (27.4)	0.865
Hyperlipidemia	31 (45.6)	51 (45.1)	1.000
Heart disease	5 (7.4)	12 (10.6)	0.602
Atrial fibrillation	2 (0.3)	5 (0.4)	0.713
Current smoking	23 (33.8)	49 (43.4)	0.214
Heavy alcohol drinker	10 (14.7)	27 (23.9)	0.183
SBP, mmHg	151.2 ± 22.5	143.8 ± 16.0	0.011
DBP, mmHg	95.6 ± 11.4	95.3 ± 11.3	0.802
Associated events			
AVS	27 (39.7)	39 (29.3)	0.525
EVS	41 (60.3)	74 (65.5)	0.525
Acute ischemic events on DWI	68 (100.0)	0 (0.0)	0.000
Recent pure ischemic events on FLAIR	0 (0.0)	65 (57.5)	0.000
TIA	0 (0.0)	39 (34.5)	0.000
Cardiac arrhythmia	0 (0.0)	5 (4.4)	0.162
Septic shock	0 (0.0)	4 (3.5)	0.298
Focal neurological sign	22 (32.4)	10 (8.8)	0.000
Large artery stenosis or occlusion on MRA	26 (38.2)	6 (5.3)	0.000
mRS score of >2 at 30-day follow-up	15 (22.1)	8 (7.1)	0.005

Data are presented as n (%) or mean ± standard deviation. AVS, acute vestibular syndrome; EVS, episodic vestibular syndrome; DWI, diffusion-weighted imaging; SBP, systolic blood pressure; DBP, diastolic blood pressure; FLAIR, fluid-attenuated inversion recovery; TIA, transient ischemic attack; MRA, magnetic resonance imaging; mRS, modified Rankin scale.

Table 3. Multivariate analysis of early predictors of central AVS/EVS caused by ischemic events.

Episodes	AVS/EVS with positive DWI signs (n = 68)	AVS/EVS with negative DWI signs (n = 113)	OR (95% CI)	p value
Large artery stenosis	26 (38.2)	6 (5.3)	0.12 (0.04–0.36)	0.000
Focal neurological sign	22 (32.4)	10 (8.8)	0.27 (0.10–0.72)	0.008

Data are presented as n (%). AVS, acute vestibular syndrome; EVS, episodic vestibular syndrome; DWI, diffusion-weighted imaging; OR, odds ratio; CI, confidence interval.

infarct in 68 (37.6%) patients. Among these patients, 42.6% of the acute infarcts were located in the territory of the ACA, 41.2% in the territory of the PCA, and the remaining 16.2% in the territory of both the ACA and PCA. Thus, the acute infarcts causing AVS and/or EVS in the ACA and PCA regions were basically balanced. Importantly, we

found that the main predilection site of infarcts causing AVS and/or EVS was the insular cortex in the ACA, followed by the posterior thalamus in the PCA; these findings indicate that these locations serve as a potential pathway for central vestibular projections. This was also confirmed in a previous study.[8]

Insular infarcts have been sporadically reported.[6,12,13] In the present series, acute small insular infarcts were the most frequent cause of AVS and/or EVS. Moreover, most lesions were located in the posterior aspect of the insular cortex. Prior research has demonstrated that the primary central vestibular cortex is located in the insular cortex.[8] Our current DWI study showed that the primary vestibular cortex may be located in the posterior region of the insular cortex. This was also confirmed in a previous electrophysiological study.[14]

Our observations indicate that AVS and/or EVS may also be caused by frontal, temporal, parietal, and occipital lobe infarcts. The cortical representation of the vestibular projections in humans is commonly assumed to be located in distinct temporal and parietal areas of the brain,[15–17] although vestibular activation has been demonstrated in the frontal lobe area.[17,18] Our current study further confirmed this viewpoint. However, we found that the occipital cortex is also an important vestibular projection area.

Few reports to date have described AVS and/or EVS caused by thalamic infarcts, but our series showed that this condition is not rare. Moreover, almost all lesions were present in the posterior thalamus. A previous study confirmed that the posterolateral thalamus is a unique relay station for vestibular input to the cortex,[19] and this was supported by our DWI study. Our current study showed that the posterior insular cortex and posterolateral thalamus are two frequent locations for stroke causing AVS and/or EVS, suggesting that they are more likely to have a distinct vestibular pathway. Furthermore, this speculation is well-supported by evidence from other studies.[19,20]

Before MRI came into use, AVS and/or EVS were very rarely considered problems associated with the ACA. Our current DWI study suggests that AVS and EVS are also symptoms of anterior circulation impairment. This problem was also found in a previous study.[21]

According to the TOAST classification standards, approximately 85% of patients with AVS and/or EVS in our series showed lacunar or small infarcts, among which ipsilateral large vessel stenosis in the ACA was common. This finding suggests that pathological changes of large vessels in the ACA are more likely to be associated with the pathogenesis of small vessel diseases. This viewpoint was confirmed in a previous study.[22]

Our current case series also showed that all patients with AVS and/or EVS in the PCA had small infarcts, and of these patients, nine had ipsilateral vertebral and basilar large vessel stenosis. Although vertebral and basilar stenosis is considered a high risk factor for posterior circulation TIA or minor stroke,[23] a recent study indicated that the presence of small infarcts in the PCA is one of the mechanisms causing severe vertigo.[24] This was also confirmed in our case series.

Moreover, the multivariate regression analysis showed that there was a significantly higher risk of AVS and/or EVS caused by acute infarcts when associated with large vessel stenosis/occlusion and focal neurological symptoms/signs, suggesting that these risk factors may contribute to acute infarcts causing AVS and/or EVS. This has also been shown in previous studies.[22,23,25] To the best of our knowledge, patients benefit from effective treatment for large vessel stenosis/occlusion with focal neurological symptoms/

signs, especially in the ACA, using early thrombolysis. Therefore, we would like to emphasize that accurately diagnosing these strokes by DWI plus MRA could potentially save lives and decrease disability through prompt intervention.

Importantly, these findings not only provide evidence for the identification of acute ischemic stroke causing AVS and/or EVS but also provide diagnostic clues for isolated AVS and/or EVS caused by ischemic event in patients with negative DWI findings. Additionally, clinicians should rule out an ACA ischemic event when a PCA ischemic event is suspected to have caused isolated AVS and/or EVS. Thus, clinicians should keep ACA stroke causing the highest-risk AVS and/or EVS in mind as well as the fact that the insular cortex is the main predilection site leading to AVS and/or EVS.

Some limitations of this study must be considered. First, although this is the first study to show that the insular cortex and posterior thalamus are the predilection sites leading to AVS and/or EVS caused by acute ischemic stroke, the results might be biased because of the retrospective nature of the analysis. However, we believe that the richest vestibular pathways in humans are in both hemispheres[8] and that the data are convincing because the data were obtained from zones with a high prevalence of cerebrovascular disease.[26] Further prospective studies are also necessary.

Second, our results confirmed that acute infarcts in the ACA or PCA can cause central AVS and/or EVS; however, nystagmus was less common in this series. This finding has two possible explanations. First, the major cause of supratentorial AVS and/or EVS in patients without nystagmus may be that the labyrinthine and oculomotor nerves are not very sensitive to pathological stimuli from supratentorial vestibular lesions. Second, even in patients with infratentorial lesions, rapid-onset nystagmus may be a transient event and therefore might not be observed because of rapid normalization of the oculomotor signs.

In addition, acute transient vestibular syndrome (i.e., EVS) has been considered to be a common cause of TIA,[4,7,25] but acute small infarcts cannot be fully excluded in patients with negative DWI findings.[27,28] Moreover, the standard location for diagnosis of acute infarcts by DWI can only confirm acute ischemic lesions. Regardless, the above conditions show that the prevalence of AVS and/or EVS caused by acute infarcts might not have been overestimated.

In conclusion, 42.6% of acute infarcts were present in the territory of the ACA and 41.2% were present in the territory of the PCA. The insular cortex is a predilection site for infarcts leading to AVS and/or EVS, followed by the posterior thalamus. This suggests that distinct vestibular pathways are present between the insular cortex and the posterolateral thalamus. The risk of AVS and/or EVS is associated with large vessel stenosis and focal neurological symptoms and signs.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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