

Acute and episodic vestibular syndromes caused by ischemic stroke: predilection sites on diffusion-weighted images

Dao-Ming Tong (✉ dmtong@xzhmu.edu.cn)

Affiliated Shuyang People' Hospital, Xuzhou Medical University

Xiao-Dong Chen

Affiliated Hospital of Xuzhou Medical College

Ye-Ting Zhou

Affiliated Hospital of Xuzhou Medical University

Tong-Hui Yang

Xuzhou Medical College Affiliated Hospital

Research article

Keywords: Acute ischemic stroke; Acute vestibular syndrome; Episodic vestibular syndrome; Diffusion-weighted images; Magnetic resonance angiography

Posted Date: May 2nd, 2019

DOI: <https://doi.org/10.21203/rs.2.9434/v1>

License:   This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Background Although acute vestibular syndrome (AVS) and episodic vestibular syndrome (EVS) are an increasingly recognized cause of acute ischemic stroke, the predilection sites of AVS/EVS caused by acute ischemic stroke still is less known. **Methods** From Mar 2014 to Mar 2016 period, we used a new approach of 11th edition of the International Classification of Diseases (ICD-11) to retrospectively enrolled patients with identified AVS/EVS events caused by acute ischemic stroke in the stroke center of tertiary teaching hospital. The patients who had positive diffusion-weighted images (DWI) lesion and MRA were analyzed. Multivariable logistic regression was used to identify the risk of stroke causing AVS/EVS. **Results** Among 181 AVS/EVS patients with ischemic stroke, 68 (37.6%) patients with acute ischemic stroke were proved by DWI. Of them, the most frequent type was EVS (60.3%); the predilection sites of stroke was in the insular (51.7%, 15/29) in the anterior circulation artery (ACA), followed by the posterior of thalamus (28.6%, 8/28) in the posterior circulation artery (PCA). The lesion on DWI showed a median diameter of 4.0mm (range,0.6-89.4). The risk of AVS/EVS in acute ischemic stroke was found in association with large vessel stenosis/ occlusion (odds ratio[OR], 0.12; 95% confidence interval [CI], 0.040-0.357), focal neurological symptom /sign (OR, 0.27; 95% CI, 0.104-0.751), and higher initial ABCD2 score (OR, 0.37; 95% CI, 0.239-0.573). **Conclusions** The predilection site of the AVS/EVS caused by acute ischemic stroke is in the insular. The risk of AVS/EVS was associated with a large vessel stenosis, focal neurological symptoms, and higher initial ABCD2 score.

Background

Although acute vestibular syndrome has been described by previous studies[1,2], a new approach of the 11th edition of the International Classification of Diseases (ICD-11) is divided patients with vertigo or dizziness into two key categories: 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, severe postural instability) is defined as acute vestibular syndrome (AVS); while 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, sudden falls) is defined as episodic vestibular syndrome (EVS). Anatomically, at any point along the vestibular pathway from the peripheral labyrinth to the central vestibular cortex, AVS/EVS may occur. Therefore, the causes of vestibular syndrome is divided into categories which include peripheral (e.g., vestibular neuronitis, acute labyrinthitis, benign paroxysmal positional vertigo [BPPV], Menière's disease) and central causes (e.g., traumatic vestibulopathy, demyelinating disease with vestibular involvement, and strokes affecting central vestibular structures). In fact, the AVS/EVS caused by impaired central vestibular structures is known as central vestibular syndrome [1,3]. Previous studies have suggested that isolated vertigo or dizziness is a symptom of posterior circulation [4,5]. But, some studies were also found a risk of signal in anterior circulating vessel involved [6,7], and the richest vestibular pathways in human beings has been demonstrated in distinct cortical and subcortical areas of both hemispheres [8]. However, few reports

have too much information about the predilection sites of central AVS/EVS. We hypothesize that AVS/EVS with positive ischemia events on diffusion-weighted images (DWI) would be more accurately represented the predisposing site of acute vestibular damage. The aims of our study were to determine whether the role of magnetic resonance imaging (MRI)-DWI combined with magnetic resonance angiography (MRA) may clarify the predilection sites of acute ischemic stroke causing AVS/EVS.

Methods

Study participants

We performed a retrospective observational study (from March 2014 to March 2016) that had consecutively recruited the adult patients with acute vertigo or dizziness and adapting an AVS/EVS definition, and they were underwent a MRI and has been in the neurologic ward and stroke center of Shuyang county hospital—the national three levels of hospital in Northern China. Here sits on a high prevalence zones of cerebrovascular disease [9], thus, the data we have collected may enough to be convincing. The study was approved by the Ethical Committee on Clinical Research of the Shuyang Hospital, Xuzhou medical University, Written informed consent was obtained from all patients or a person who had been designated to give consent on admission of the patient.

For this study, The inclusion criteria of the AVS/EVS were as follows: (1) acute onset of dizziness/vertigo, adapting the standard definition of AVS/EVS published by the 11th edition of the International Classification of Diseases (ICD-11); (2) within 24-72 hours from the symptoms onset, an ischemic stroke event was confirmed by DWI or FLAIR; (3) no previous history of recurrent dizziness/vertigo. Exclusion criteria were included the peripheral AVS/EVS (e.g., BPPV, Menière's disease), migrainous vertigo, spontaneous intracerebral hemorrhage, vertebralarterial type cervical spondylopathy, demyelinating disease, drug-induced causes, and other causes of non-ischemic stroke.

Of 502 adult patients who had acute vertigo or dizziness and adapting an AVS/EVS definition and underwent a MRI, after excluding 186 patients with peripheral causes (e.g., BPPV etc), 71 migrainous vertigo, 31 intracerebral hemorrhage, 14 vertebralarterial type cervical spondylopathy, 10 demyelinating disease, 6 drug- induced causes, and 3 other causes of non- ischemic stroke, finally, 181 patients who met the inclusion criteria were included in present study. According to a confirmation of increased signal on DWI for acute infarcts diagnosis, the study population was divided into two groups: AVS/EVS with positive DWI and negative DWI groups.

MRI protocol

MRI was performed with 1.5-T equipment (Siemens AG, Munich, Germany) in all patients. MRI sequences included conventional T2, T1, FLAIR, and DWI on an axial view, from medulla to cortex, at 5-mm section thickness. The MRA was used for 3D-TOF imaging on axial scan, the interval was -0.6 mm, each slice thickness of 1.1 mm. the range of the brain MRA imaging included the main branches and the trunk in the anterior circulations and posterior circulations, as well as the entire Willis artery cycle.

Assessment of study patients

We referred to the Oxford community stroke project standard, the diagnosis of ischemic infarction in a vascular region of the brain is divided into two regions: anterior circulation artery (ACA) and the posterior circulation artery (PCA). Symptomatic carotid artery or vertebral basilar artery stenosis was defined as $\geq 50\%$ diameter reduction of in a location of the carotid artery or vertebral basilar artery that was considered likely to be responsible for the any acute infarct or the localization of the clinical symptoms. Based on the trial of ORG 10172 in acute stroke treatment for classification of subtypes in acute ischemic stroke[10], the lesion on brain MRI-DWI is greater than 15mm in diameter as the large infarcts, whereas ≤ 15 mm in diameter as the small infarcts(lacunar infarcts).

Clinically, Isolated AVS/EVS is referred to its caused by pure vestibular structure lesion; while non isolated AVS/EVS is referred to its caused by a vestibular structure lesion and with focal neurological symptom/sign. The ABCD 2 score (age, blood pressure, clinical features, duration, and diabetes mellitus) was also calculated for each patients, which is a risk score for patients with TIA. We defined a TIA as an episode < 24 hours of neurologic dysfunction (ABCD2 >3 scores) caused by focal brain, spinal cord, or retinal ischemia, without recent and new infarction.

Data Collection

The initial diagnosis of MR is determined by a highly qualified radiologist. MRI-DWI infarct diameter (mm) in each MRI slice was measured by a highly qualified neurologist, and detailed the records of each patient's number and position of lesions. The images of MRA were measured, and the location and the abnormal condition of blood vessels were registered by same highly qualified neurologist. At the same time, we also enrolled the patient's other information, including gender, age, history of hypertension, history of diabetes mellitus, atrial fibrillation or history of heart disease, drinking, smoking, clinical symptoms, ABCD 2 score, and laboratory examination. For outcome analyses, the modified Rankin Scale (mRS) score (scores range from 0 to 6, no symptoms=0, slight symptoms=1, restriction=2, slight disability=3, moderate disability=4, severe disability=4-5, death=6) follow-up information at 30-days was gathered and assessed retrospectively by a neurological specialist.

Statistical analysis

The measurement data of all patients in this study were expressed as mean plus or minus standard deviation (mean + SD), median, or percentage, and the count data were measured by t test. Multivariable logistic regression was used to determine the risk factors of stroke causing AVS/EVS. Data analysis using SPSS17.0 Version (SPSS Corporation, Chicago, IL, USA).

Results

During the study period, of 181 patients with an adapting AVS/EVS caused by ischemic events, 68 with positive DWI ischemic events (acute infarcts) and 113 negative DWI ischemic events (e.g., recent ischemic

events on FLAIR, TIA, septic shock, cardiac arrhythmia) were observed by MRI and MRA (in 98.5% and 90.3%, respectively). Univariate analysis revealed a significantly increased risk of ischemic event in patients with positive DWI and the following risk factors: age > 60 years (60.3% vs. 44.2%, $p=0.048$), SBP (151.2 ± 22.5 vs. 143.8 ± 16.0 , $p=0.011$), initial ABCD 2 score (4.1 ± 1.0 vs. 3.2 ± 0.8 , $p=0.000$), focal neurological symptom/sign (32.4% vs. 8.8%, $p=0.000$), and large vessel stenosis (38.2% vs. 5.3%, $p=0.000$) (Table 1). However, the other risk factors did not differ between the groups ($P > 0.05$). The results of multivariate regression analysis showed that only the large vessel stenosis or occlusion (OR, 0.12; 95% CI, 0.040-0.357), focal neurological symptom/sign (OR, 0.27; 95% CI, 0.140-0.715), and higher initial ABCD2 score (OR, 0.37; 95% CI, 0.239-0.573) was an independently risk factors of stroke in AVS/EVS patients with positive acute ischemic events on DWI (Table 2).

Among 68 (37.6%) patients with positive DWI causing central AVS/EVS, from the emergency to the hospital admission time was the shortest for half an hour, the longest 14 days, the median time of 1.5 days. The median time from the onset to MRI-DWI was 1.5 days (range: 0.2-14.8d). Imaging change and clinical features of 68 patients with AVS/EVS caused by acute ischemic stroke are shown in the table 3.

Pure ACA region infarcts was in 29 (42.6%) cases. The most frequent location (51.7%, 15/29) was in the insular. Of them, 14 patients had unilateral insular lesion and the majority of lesions were located in the posterior of the insular. (Figure 1) Moreover, the non isolated insular infarcts were found over a half of these patients (8/15).

The other cerebral lobe infarcts in the ACA were also common (14/29; 48.3%) (Figure 2), including the frontal lobe in 4 cases, parietal lobe in 3 cases, frontal temporal parietal in 2 cases, temporal parietal lobe in 2 cases, temporal lobe in 1 cases, periventricular in 2 cases. In the ACA, the ipsilateral large vessel stenosis/occlusion confirmed by MRI was in 12 (46.2%) cases, including the ipsilateral middle cerebral artery stenosis/occlusion in 6 cases, the ipsilateral internal carotid artery occlusion in 4 cases, the ipsilateral anterior cerebral artery occlusion in 2 cases.

The pure posterior circulation infarcts was in 28 (41.2%) cases. DWI showed that 7 cases impaired thalamic vestibular structure was usually limited to the posterolateral thalamus (Figure 3). All 8 patients was unilateral lesions, of these, isolated lesion was only present in 2 patients.

The other cerebral infarcts in the PCA included the pons brain in 7 cases, cerebellum in 6 cases, occipital lobe in 4 cases, and midbrain in 3 cases. In the posterior circulation infarcts, 9 (34.6%) cases had ipsilateral vertebral basilar artery stenosis/occlusion confirmed by MRA.

The ACA associated PCA were involved in 11 (16.2%) cases, including the parietal lobe and cerebellum in 3 cases, temporal parietal occipital lobe in 2 cases, parietal lobe and medullar in 1 (9.1%) case, cingulum in 2 cases, and other 3 cases. Among them, there were 5 cases with large vascular stenosis/occlusion, including the unilateral middle cerebral artery in 3 cases, 1 case in the unilateral superior cerebella artery, and 1 case in the unilateral vertebral artery.

DWI showed that the median diameter of infarct was 4.0mm (range: 0.6-89mm). The patients with small infarcts confirmed by DWI were in 58 (85.3%) cases, and there were only 18(31.0%) cases with ipsilateral large vessel stenosis or occlusion confirmed by vascular imaging (Figure 4).

Infarct lesion was greater than 15mm in 10 patients, of which MRA showed ipsilateral large vessel stenosis or occlusion in 8 cases. The other 2 cases were unknown. TOAST etiology of 68 patients was classified (Table SI).

For outcome analyses, 15 of the 68 AVS/EVS patients who had positive DWI events had a slight-moderate disabling (mRS 3-4 score) at 30 days follow-up after the initial event compared with 8 of the 113 AVS/EVS patients who had negative DWI events (22.2% vs.7.1%, $p=0.001$) (Figure 5). No patients died at 30-day follow-up.

Discussion

Our series of patients by brain DWI study found that 68 (37.6%) patients with AVS/EVS were caused by acute ischemic infarcts. The predilection sites of infarcts causing AVS/EVS is in the insular in the ACA, followed by posterior thalamus in the PCA. supporting that these locations have to exist a potential pathway of the central vestibular projections. This has also been confirmed by the previous study [8].

Insular infarcts have been sporadic cases reported[6,11,12]. In our series, the acute small insular infarcts were the most frequent cause of AVS/EVS. Moreover, the majority of lesions were located in the posterior of the insular. Prior research has demonstrated that the primary central vestibular cortex is located in the insular cortex [8]. Our current DWI study showed that primary vestibular cortex may be at the posterior of the insular. This has also been confirmed by previous electrophysiological study [13].

Our observations indicated that AVS/EVS may also be caused by frontal, temporal, parietal, and occipital lobe infarcts. The cortical representation of the vestibular projections in human beings is commonly assumed to be located in distinct temporal and parietal area of the brain [14-16], although vestibular activation has been demonstrated in frontal lobe area [16,17]. Our current study further confirmed the above view. However, we found that the occipital cortex is also an important vestibular projections area.

Few have reported the thalamic infarcts causing AVS/EVS in the previous literature. Our series showed that thalamic infarcts causing AVS/EVS were not rare. Moreover, almost all lesions were present in the posterior thalamus. Previous study has confirmed that the posterolateral thalamus is a unique relay station for vestibular input to the cortex [18], and this has been supported by our study of DWI. Our current study showed that the posterior insular and posterolateral thalamus was two frequent locations for stroke causing AVS/EVS, suggesting that here is more likely to have a distinct vestibular pathway. Furthermore, this speculation is well-supported by evidence from other studies [18,19].

To our best acknowledge, before the MRI has not emerged, AVS/EVS is very rare to consider the problem of ACA. Our current DWI study suggests that AVS/EVS is also a symptom of anterior circulation impaired.

This problem has been found by previous study [20].

According to TOAST classification standards, in our series, approximately 85% of our patients with AVS/EVS showed lacunar or small infarcts, in which the ipsilateral large vessel stenosis in the ACA was common. This finding suggests that the pathological changes with large vessel in the ACA are more likely to be associated with the pathogenesis of small vessel diseases. This point has been confirmed by a previous research [21].

Our current case series also found that all patients with AVS/EVS in the PCA were small infarcts, and having 9 patients with ipsilateral vertebral and basilar large vessel stenosis. Although vertebral and basilar stenosis is supposed to be a high risk factor for posterior circulation TIA or minor stroke [22], a recent study indicated that the small infarcts in the PCA is one of the mechanisms causing severe vertigo [23]. This has also been confirmed by our case series.

Moreover, multivariate regression analysis showed that there was a significantly higher risk of AVS/EVS caused by acute infarcts when associated with large vessel stenosis/occlusion, focal neurological symptom/sign, and higher initial ABCD2 score, suggesting these risk factors may be contributed to acute infarcts causing AVS/EVS, which were reported by previous studies [21,22,24,25].

However, the limitation of our study still is conceded. First, our results confirmed that acute infarcts in the ACA or PCA causing a central AVS/EVS, but that, unfortunately, is less common than the nystagmus in this series. To explain this issue there are two possible facts: the first should be that the majority of supratentorial AVS/EVS patients without a nystagmus may be due to the labyrinthine and oculomotor vestibular nuclei unperceived to the pathological stimuli from supratentorial vestibular lesions; and the second that, even lesion in infratentorial, rapid onset nystagmus may also be a transient event, therefore it did not be caught due to the rapid normalization of the ocular motor signs.

Second, acute transient vestibular syndrome (i.e. EVS) has been considered to be a common cause of TIA [4,7,24], but negative DWI may also not be fully excluded acute small infarcts [26,27]. Moreover, we have to admit that the location of standard for diagnosis of acute infarcts is only a confirmation of acute ischemic lesion with DWI. In addition, we excluded the patients who were migrainous vertigo in which this information was unavailable because of present in migrainous rather than isolated vertigo. No matter how, above conditions showed that the prevalence of AVS/EVS caused by acute infarcts might be underestimated.

In conclusion: The insular is the predilection sites leading to AVS/EVS caused by small ischemic stroke, followed by the posterior thalamus, suggesting that there is distinct vestibular pathways between the insular and the posterolateral thalamus. The risk of AVS/EVS was associated with a large vessel stenosis, focal neurological symptom/sign, and higher ABCD 2 score.

Abbreviations

AVS: acute vestibular syndrome; **EVS:** episodic vestibular syndrome; **DWI:** Diffusion-weighted images; **MRA:** Magnetic resonance angiography; **ABCD2:** age, blood pressure, clinical features, duration, and diabetes mellitus.

Declarations

Acknowledgment

We would like to thank everyone who contributed to the study, namely the staff of the ICU who facilitated the access to patient records and the nurses and physicians for reviewing medical records.

Funding

From Clinical Key Specialty Construction Project (201400016), the Medical Research Council, affiliated Shuyang Hospital of Xuzhou Medical University, Jiangsu, China.

Availability of data and materials. Please contact author for data requests

Competing interests

All authors declare that no conflicts of interest exist.

Authors' contributions

Conceived and designed the experiments:DMTand XDC.

Performed the experiments:DMT,XDC.

Analyzed the data:DMT,XDC,YTZ and THY.

Contributedreagents/materials/analysistools:DMT XDC YTZ,THY.

Wrote the paper:DMT.

Agree with manuscript results and conclusions: DMT, XDC, YTZ, THY.

Ethics approval and consent to participate

The study was approved by the ethical committee on clinical research of the Affiliated Shuyang Hospital of Xuzhou Medical University and Affiliated Pingxiang Hospital of the Southern Medical University, and written informed consent was obtained from the patients or their family.

Consent for publication

Not applicable.

References

1. Tamutzer AA, Berkowitz AL, Robinson KA, et al. Does my dizzy patient have a stroke? A systematic review of bedside diagnosis in acute vestibular syndrome. *CMAJ* 2011;183:E571-E592.
2. Hotson JR, Baloh RW. Acute vestibular syndrome. *N Engl J Med* 1998 ; 339:680-685.
3. Kim HA, Lee H. Recent advances in central acute vestibular syndrome of a vasculae cause. *J Neurol Sci.* 2012 15; 321 (1-2):17-22.
4. Paul NL, Simoni M, Rothwell PM. Transient isolated brainstem symptoms preceding posterior circulation stroke: A population-based study. *Lancet Neurol*, 2013;12:65-71.
5. Searls DE, Pazdera L, Korbel E, Vysata O, Caplan LR. Symptoms and signs of posterior circulation ischemia in the New England Medical Center posterior circulation registry. *Arch Neurol.* 2012;69: 346.
6. Cereda C, Ghika J, Maeder P, Bogousslavsky J. Strokes restricted to the insular cortex. *Neurology.* 2002 24; 59:1950-1955.
7. Okroglic S, Widmann CN, Urbach H, Scheltens P, Heneka MT. Clinical symptoms and risk factors in cerebral microangiopathy patients. *PloS one.* 2013; 8:e53455.
8. Duque-Parra JE. Perspective on the vestibular cortex throughout history. *Anat Rec B New Anat.* 2004; 280:15-9.
9. Xu G, Ma M, Liu X, Hankey GJ. Is there a stroke belt in China and why? *Stroke.* 2013; 44:1775-1783.
10. Adams HP Jr, Bendixen BH, Kappelle LJ, Biller J, Love BB, Gordon DL. Classification of subtype of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. *Stroke.* 1993;24:35-41.
11. Brandt T, Bötzel K, Yousry T, Dieterich M, Schulze S. Rotational vertigo in embolic stroke of the vestibular and auditory cortices. *Neurology* 1995;45:42-44.
12. Ahn BY, Bae JW, Kim DH, Choi KD, Kim HJ, Kim EJ. Pseudo-vestibular neuritis associated with isolated insular stroke. *J Neurol* 2010;257:1570-2.
13. Lopez C, Blanke O, Mast FW. The human vestibular cortex revealed by coordinate-based activation likelihood estimation meta-analysis. *Neuroscience.* 2012 14;212:159-79.
14. Bucy PC. Vertigo with diseases of the central nervous system. *Arch Otolaryng* 1967;85:91-92.
15. Toth LI, Assad JA. Dynamic coding of behaviourally relevant stimuli in parietal cortex. *Nature* 2002;415:165-168.

16. Dieterich M, Bense S, Lutz S, Drzezga A, Stephan T, Bartenstein P, et al. Dominance for vestibular cortical function in the non-dominant hemisphere. *Cereb Cortex*. 2003;13(9):994-1007.
17. Kluge M, Beyenburg S, Fernandez G, Elger C. Epileptic vertigo: evidence for vestibular representation in human frontal cortex. *Neurology* 2000;55:1906-1910.
18. Dieterich M, Bartenstein P, Spiegel S, Bense S, Schwaiger M, Brandt T. Thalamic infarctions cause side-specific suppression of vestibular cortex activations. *Brain*.2005;128:2052-67.
19. Ray JP, Price JL. The organization of the thalamocortical connections of the mediodorsal thalamic nucleus in the rat, related to the ventral forebrain-prefrontal cortex topography. *J Comp Neurol*.1992. 8;323(2):167-97.
20. Anagnostou E, Spengos K, Vassilopoulou S, Paraskevas GP, Zis V, Vassilopoulos D. Incidence of rotational vertigo in supratentorial stroke: a prospective analysis of 112 consecutive patients. *J Neurol Sci*.2010, 15;290(1-2):33-6.
21. Brisset M, Boutouyrie P, Pico F, Zhu Y, Zureik M, Schilling S, et al. Large-vessel correlates of cerebral small-vessel disease. *Neurology*; 2013;80:662-669
22. Marquardt L, Kuker W, Chandratheva A, et al. Incidence and prognosis of $\geq 50\%$ symptomatic vertebral or basilar artery stenosis: prospective population-based study. *Brain* 2009, 132;982-988.
23. Saber Tehrani AS, Kattah JC, Mantokoudis G, et al. Small strokes causing severe vertigo: frequency of false-negative MRIs and nonlacunar mechanisms. *Neurology*.2014; 8;83:169-73.
24. 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.
25. Navi BB, Kamel H, Shah MP, et al. Application of the ABCD2 score to identify cerebrovascular causes of dizziness in the emergency department. *Stroke*. 2012;43:1484-1489.
26. Doubal FN, Dennis MS, Wardlaw JM. Characteristics of patients with minor ischaemic strokes and negative MRI: a cross-sectional study. *J Neurol Neurosurg Psychiatry*. 2011; 82(5): 540-2. doi: 10.1136/jnnp.2009.190298. Epub 2010 Jun 27
27. Winbeck K, Bruckmaier K, Etgen T, von Einsiedel HG, Röttinger M, Sander D. Transient ischemic attack and stroke can be differentiated by analyzing early diffusion-weighted imaging signal intensity changes. *Stroke*. 2004; 35: 1095-1099

Tables

Due to technical limitations, Table(s) 1-3 are only available as a download in the supplemental files section.

Figures

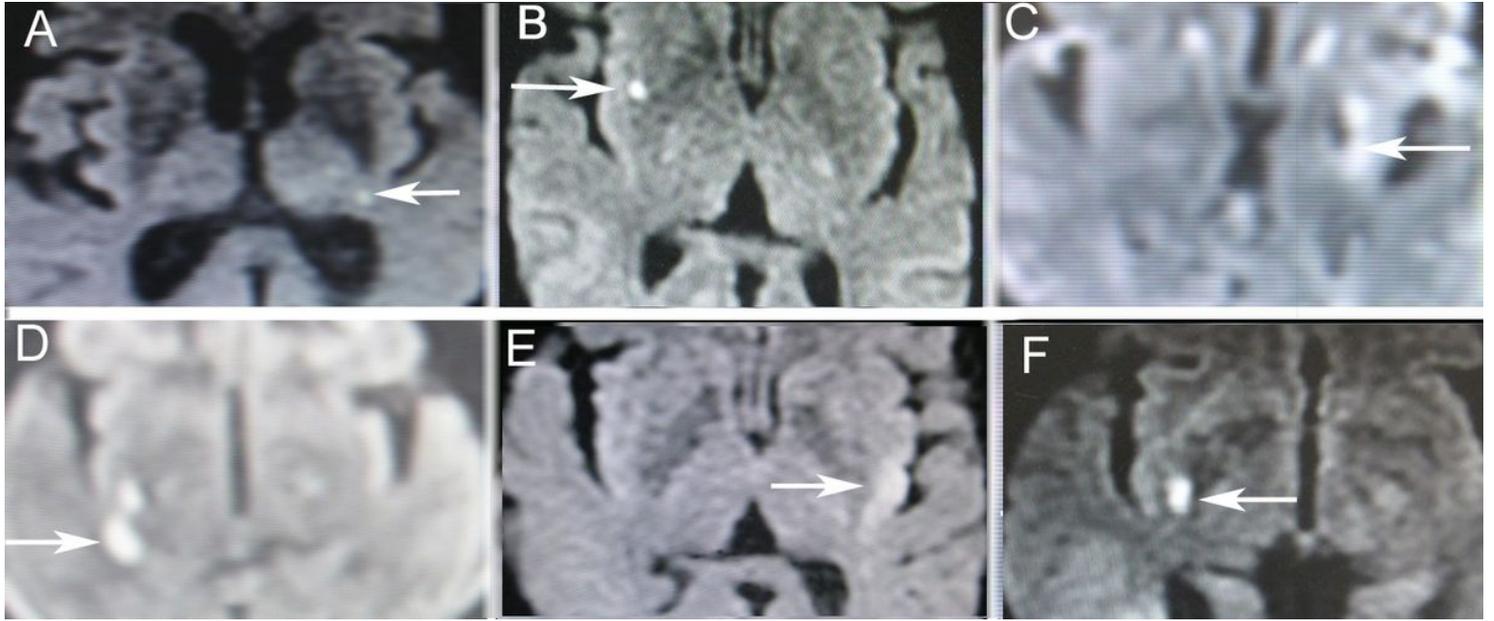


Figure 1

MRI-DWI acute insular infarcts in 15 of 68 patients with central AVS/EVS. Here, only select 6 images representative of different topographical region involved are shown. The median-posterior insular infarcts (A, B, C, and F, arrows) show the featuring of forming small, and elliptical infarcts. Figure 1 (D and E, arrow) presents the dorsal insular infarct.

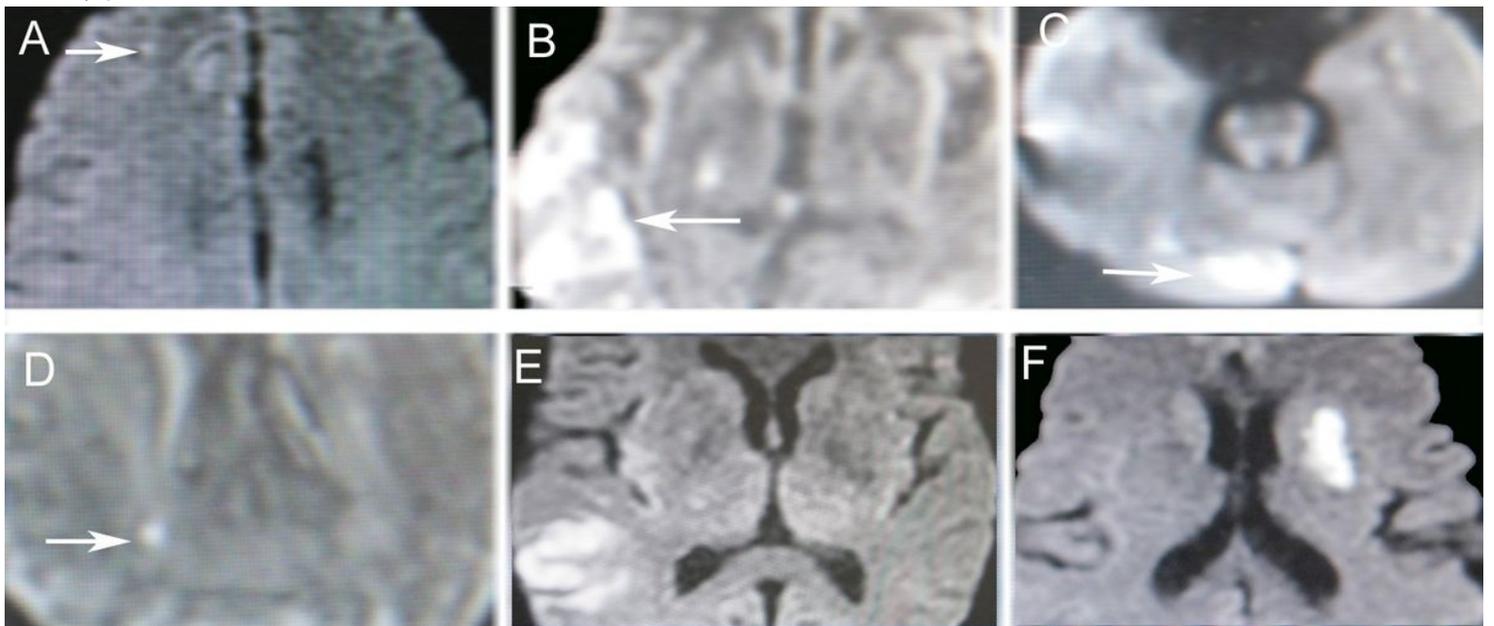


Figure 2

MRI-DWI acute cerebral lobe infarcts in 14 of 68 patients with central AVS/EVS. Only selected 6 images representative of different topographical region involved are shown. Fig.2 (A, arrow) represent a frontal

lobe infarct. Fig2 (B,arrow) represent the temporal parietal lobe infarct. Fig2 (C and D,arrow) represent the occipital lobe infarct.Fig.2 (E, arrow) represent a parietal lobe infarct.Fig.2 (F, arrow) represent a left frontal lobe infarct and spreading to the regions of the insular lobe.

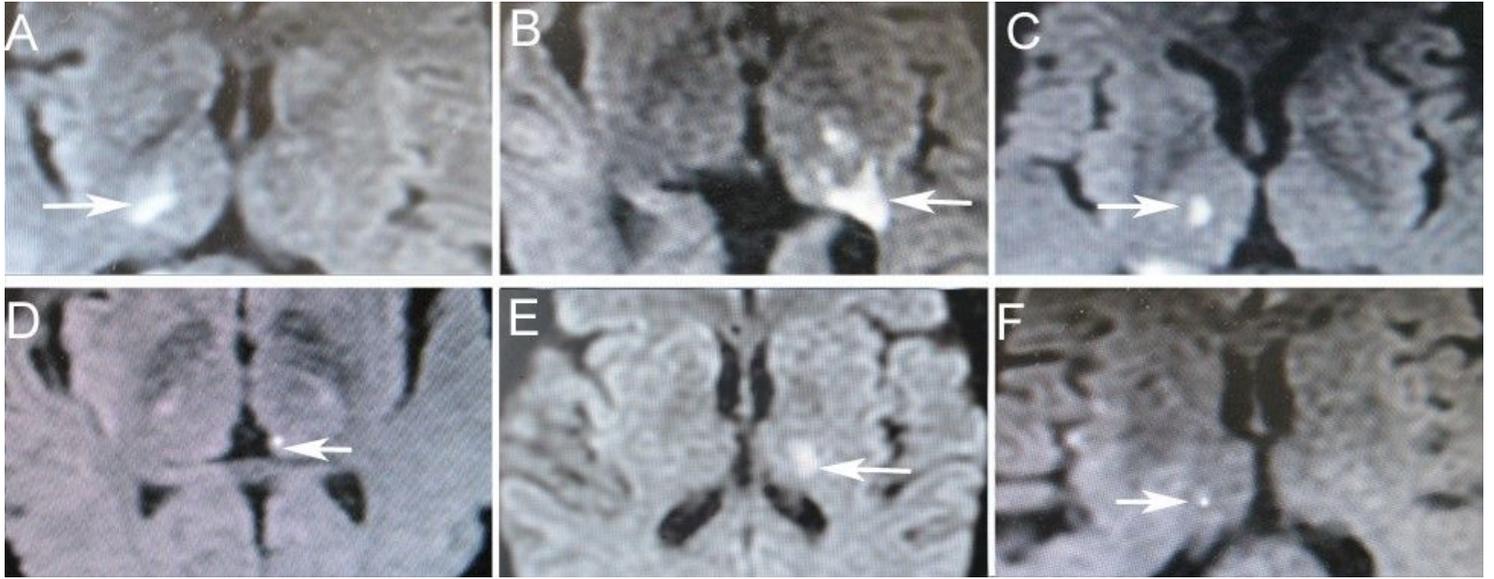


Figure 3

MRI-DWI showing acute thalamic vestibular infarcts in 8 of 68 patients with central AVS/EVS. Here, select 6 images representative of different topographical region involved are shown. Thalamic vestibular structure infarcts are usually limited to the posterolateral thalamus (A,B,C,E, and F, arrows). Only 1 patient with vestibular lesion is limited to the dorsal of the thalamus (D, arrows).

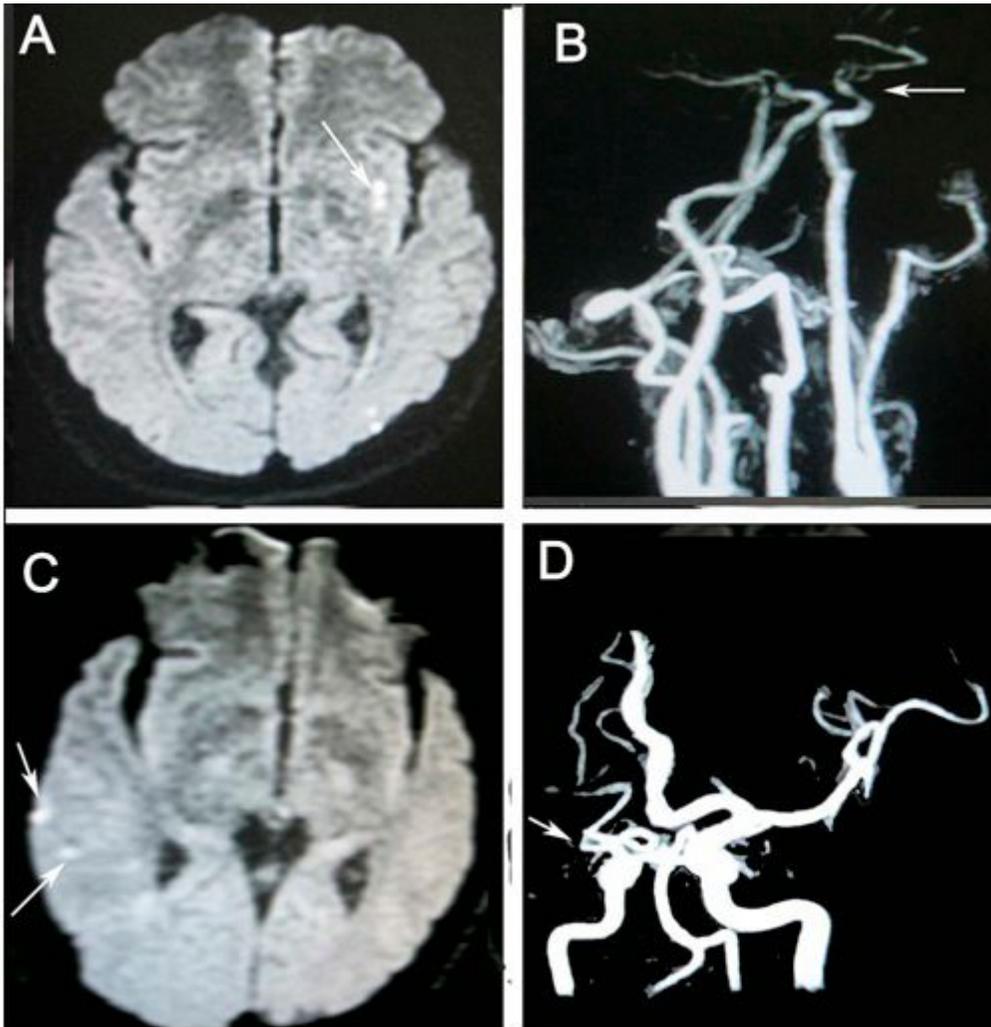


Figure 4

DWI and vascular imaging of acute infarction in the vestibular throwing structure of the anterior circulation. A 40-year-old male with AVS and slightly slurred speech for 4 days, acute small infarct in the left insula (A, arrow) are visible on the head MRI-DWI; CT vascular imaging display in the beginning of the left internal carotid artery stenosis (B, arrows). A 55-year-old male with EVS for 1 day, the first MRI-DWI shows the right temporal lobe (C, arrow) acute small infarction; MRA shows the right side of the middle cerebral artery occlusion (D, arrow).

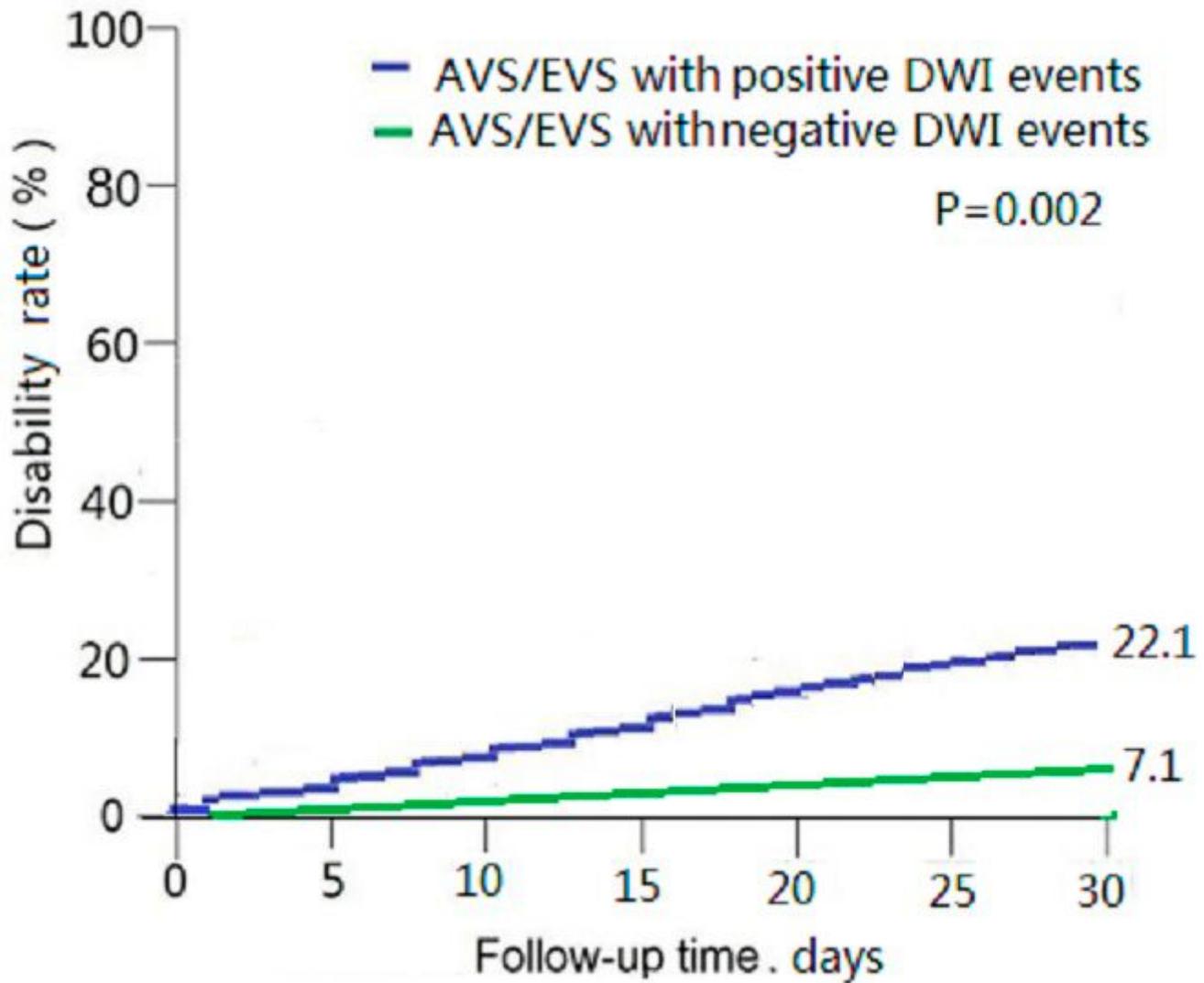


Figure 5

Risk for disabling events in the first 30 days after initial event for patients with positive DWI events had significantly higher disabling rate than patients with negative DWI events (22.1% vs.7.1% P = 0.002).

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [Table2.pdf](#)
- [Table3.pdf](#)
- [renamedd47c3.doc](#)
- [Table1.pdf](#)