Neurology® Clinical Practice

Isolated vestibular syndrome in posterior circulation stroke

Frequency and involved structures

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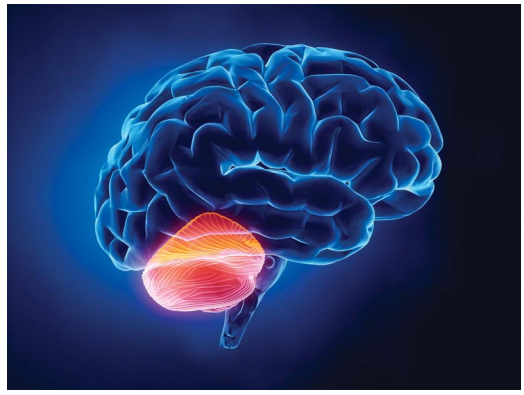
Summary

Dizziness/vertigo is a common symptom of posterior circulation stroke and usually accompanies other neurologic symptoms and signs. Although strokes involving the brainstem or cerebellum may produce isolated vestibular syndrome (isolated vertigo or imbalance), the overall frequency and involved structures of isolated vestibular syndrome in the posterior circulation stroke remain uncertain. Isolated vestibular syndrome occurs in approximately 25% of the patients with posterior circulation stroke, and mostly involves the cerebellum, inferior or superior cerebellar peduncles, and caudal lateral or rostral dorsolateral medulla. The occasional negative neuroimaging in patients with acute isolated vascular vertigo highlights the importance of appropriate bedside evaluation in acute vestibular syndrome.



Dizziness/vertigo is one of the most common symptoms of posterior circulation stroke.[1,2] Recent prospective studies using a large database reported dizziness as a presenting symptom in 47%–75% of patients with a posterior circulation stroke.[2,3] Even though dizziness/vertigo in cerebrovascular disorders usually accompany other

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neurologic symptoms and signs, recent advances in clinical neurotology and neuroimaging have led to a consensus that strokes involving the brainstem or cerebellum produce isolated vestibular syndrome (isolated vertigo or imbalance) more often than previously believed.[4] Transient isolated vertigo is also one of the most common manifestations of vertebrobasilar insufficiency.[5]

The cerebellum is known to be responsible for isolated vestibular syndrome.[6–9] A study showed that 25% of patients who presented to an emergency medical setting with isolated vertigo have a cerebellar infarction in the territory of the medial branch of the posterior inferior cerebellar artery (PICA). Furthermore, about 11% of patients with a cerebellar infarction in the PICA territory presented with isolated vertigo.[7] In the brainstem, earlier reports have described several neural structures as responsible for isolated vestibular syndrome,[6] which include the vestibular nucleus and root entry zone of the eighth nerve in the pontomedullary junction,[10–13] rostral dorsolateral or caudal lateral medulla,[14,15] paramedian pontine tegmentum,[16] midbrain,[17] and cerebellar peduncles.[18] However, since previous reports on isolated vestibular syndrome are mostly limited to anecdotal case reports and specific subtypes of posterior circulation ischemia, the overall frequency and involved structures of isolated vestibular syndrome in the posterior circulation infarctions remain to be determined.

This study aimed to determine the frequency and involved structures of acute isolated vascular vertigo by analyzing the data of 132 prospectively recruited consecutive patients with posterior circulation infarctions in a referral Stroke Center.

METHODS

Subjects

We recruited 132 consecutive adult patients with acute to subacute infarctions (<14 days from the symptom onset) involving the posterior circulation territory at the Stroke Center of Pusan National University Hospital in Korea from July 2011 to August 2012. The infarctions were documented by CT or MRI of the brain during the acute to subacute phase. All patients underwent bedside neurologic and neuro-otologic examinations by a stroke neurologist and a neuro-otologist. The patients included 97 men (73%) and 35 women (27%) with ages ranging from 17 to 89 years (mean ± SD = 68.3 ± 11.0 years). The majority were examined within 48 hours of symptom onset (69/132, 72.7%), and the median time from the symptom onset to first examination was 16.5 hours (range 30 minutes to 14 days).

We defined isolated vestibular syndrome from posterior circulation infarctions as follows: (1) rapid onset of vertigo or imbalance, and no other neurologic symptoms; (2) no general neurologic signs such as mental status abnormality, facial palsy, limb weakness, sensory loss, visual field defects, limb ataxia/dysmetria, dysarthria, dysphonia, dysphagia, and other cranial neuropathy, or obvious ocular motor signs that included ocular motor nerve palsies, internuclear ophthalmoplegia, and gaze palsy; (3) infarcts in the territories of the posterior circulation documented on CT or MRI. We excluded patients with additional infarctions in the anterior circulation territories.

Bedside neuro-otologic evaluation included examinations for spontaneous, gaze-evoked, head-shaking, and positional nystagmus, and bedside horizontal head impulse without using video-based equipment, prism cross-cover test for ocular alignment, horizontal smooth pursuit and saccades, and assessment for balance.[9,19,20] Imbalance was graded from 0 to III based on the following criteria: grade 0 (normal), able to stand on tandem Romberg with the eyes open for 3 seconds; grade I (mild), unable to stand on tandem Romberg with the eyes open at least for 3 seconds; grade II (moderate), unable to stand on Romberg with the eyes open at least for 3 seconds; grade III (severe), unable to stand or sit without support.[9]

Radiologic evaluation

All patients with general neurologic or obvious ocular motor signs had brain imaging. In patients with isolated vestibular syndrome, we performed an imaging when the patients had profound craniocervical pain, positive HINTS (the presence of normal head impulse test,

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Involved structures were assessed by a neurologist and neuroradiologist without knowing the clinical and laboratory findings of the patients.

direction-changing nystagmus, or skew deviation), or severe postural imbalance (grade III). The reference of standard for a stroke diagnosis was a confirmation of acute to subacute stroke by neuroimaging, mostly MRIs with diffusion-weighted imaging (DWI). Almost all the patients (130/132, 98.5%) underwent stroke protocol MRIs at the time of admission, and the remaining 2 received brain CT due to a pacemaker. Brain CT in those patients showed an unequivocal cerebellar stroke. Magnetic resonance angiogram was performed in 129 patients (97.7%). Initial imaging occurred within 6 hours from the bedside evaluation in all patients. Brain images were obtained within 72 hours of symptom onset in 84% and within 96 hours in 90%. The remaining 13 patients had the imaging between 5 to 14 days from the symptom onset. Ten patients with initially negative neuroimaging repeated MRIs due to unexplained or persistent signs suggestive of posterior circulation strokes.

Determination of the involved structures and their vascular territories

The areas of infarctions were determined clinically and with brain imaging. Involved structures were assessed by a neurologist and neuroradiologist without knowing the clinical and laboratory findings of the patients. The intracranial posterior circulation territories were determined based on magnetic resonance anatomic templates.[21]

We also classified the involved territories into proximal, middle, distal, and multiple based on a previous report on posterior circulation stroke.[2] The proximal territory includes the regions supplied by the intracranial vertebral artery and the PICA. The middle territory comprises the structures supplied by the anterior inferior cerebellar artery (AICA) and branches of the basilar artery (BA) up to the superior cerebellar artery (SCA). The distal territory corresponds to the areas supplied by the rostral BA, SCA, posterior cerebral artery (PCA), and their penetrating branches. We analyzed the frequency of isolated vestibular syndrome in each vascular territory.

In patients with isolated vestibular syndrome due to cerebellar infarction, we plotted the lesion sites using an atlas[21] and coded the lesion location as the inferior cerebellar hemisphere, tonsil, pyramid, nodulus and uvula, flocculus, and dentate nucleus.

Statistics

χ^2 tests were used to compare the frequency of isolated vestibular syndrome among the groups with 3 different vascular territories. Statistical analyses were conducted using SPSS 15.0 (SPSS/PC, Chicago, IL), and p values <0.05 were considered significant.

Standard protocol approvals, registrations, and patient consents

All experiments followed the tenets of the Declaration of Helsinki, and the institutional review boards of Pusan National University Hospitals approved this study. Written informed consent was obtained from all participants.

RESULTS

Involved territories

A total of 111 patients (84.1%) had single territorial infarcts on brain imaging. The most frequent location was the proximal territory (44/132, 33.3%), followed by the distal in

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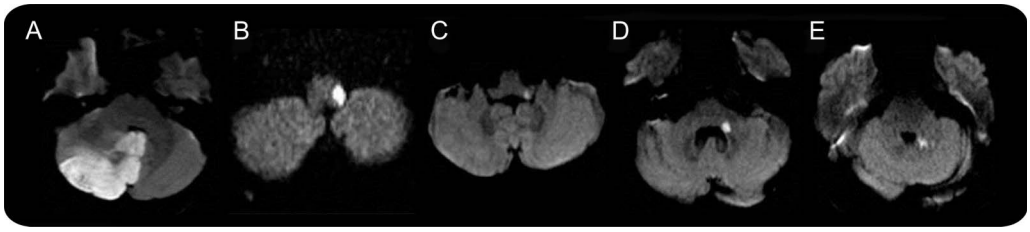
Figure 1 MRIs of the representative patients with isolated vestibular syndrome



The lesions mostly involve the cerebellum in the territory of medial posterior inferior cerebellar artery (A), caudal lateral (B) or rostral-dorsolateral medulla (C), and inferior (D) or superior cerebellar peduncles (E).

35 (26.5%), and the middle territory in 32 (24.2%) patients. Twenty-one patients (15.9%) had infarctions involving more than 1 territory. Thirty-two patients (23.2%) had infarctions restricted to the cerebellum, which include 27 in the PICA (84%), 1 in the SCA, and 4 in the multiple vascular territories (3 in the PICA and SCA, and 1 in the PICA, AICA, and SCA).

Frequency of isolated vestibular syndrome

Of the 132 patients with posterior circulation infarctions, we identified 34 (25.8%) patients with isolated vestibular syndrome (table e-1 at Neurology.org/cp). Most of the patients with isolated vestibular syndrome had infarctions involving the proximal territory (27/34, 79.4%). The frequency of isolated vestibular syndrome was 61.4% (27/44) in the proximal, 3.1% (1/32) in the middle, 5.7% (2/35) in the distal, and 19.0% (4/21) in the multiple territory groups. The frequency of isolated vestibular syndrome was highest in the proximal group ($p < 0.01$).

MRI lesions in isolated vestibular syndrome

In the 34 patients with isolated vestibular syndrome, the most common site of infarction was the cerebellum (23/34, 67.6%, figure 1). Other structures included the cerebellar peduncles (3 inferior and 2 superior; 5/34, 14.7%), caudal lateral (4/34, 11.8%) or rostral-dorsolateral medulla (2/34, 5.9%), paramedian pons (1/34, 2.9%), and occipital lobe (1/34, 2.9%). One patient (patient 28) initially presented with the features of right peripheral vestibulopathy, but with profound imbalance, and the initial MRIs were normal. On the next day, he developed right ataxia and hearing loss, and follow-up imaging disclosed an acute infarction in the right middle cerebellar peduncle (MCP).

The most commonly involved vascular territories for isolated vestibular syndrome was the PICA (27/34, 79.4%, figure 1). Other vascular territories included the SCA (n = 1), AICA (n = 1), combined PICA, AICA, and SCA (n = 1), combined PICA and SCA (n = 2), combined BA and PICA (n = 1), and PCA (n = 1). Of the 23 patients with isolated vestibular syndrome due to cerebellar infarction, 21 (91.3%) had an infarction in the territory of medial PICA (table e-1). Lesion analysis in 23 patients with isolated vestibular syndrome due to cerebellar infarction revealed that the inferior hemisphere was affected in all (100%), followed by the tonsil in 17 (73.9%), the pyramid in 9 (39.1%), the uvula and nodulus in 8 (34.8%), the dentate nucleus in 3 (13.0%), and the flocculus in only 1 (4.3%).

Initial neuroimaging including DWI was falsely negative in 10 (10/132, 7.6%) patients with posterior circulation infarction, and in 6 (6/34, 17.6%) with isolated vestibular syndrome. Negative images were mostly obtained within 24 hours (70%) from the symptom onset. However, 3 patients showed initially negative imaging even more than 24 hours after the symptom onset. Follow-up MRIs, performed with an average interval of 38 hours (range 3–135 hours), revealed infarctions in the caudal lateral medulla (n = 3), rostral dorsolateral medulla and inferior cerebellar peduncle (ICP, n = 1), MCP (n = 1), or ICP and superior cerebellar peduncle (SCP, n = 1) in 6 patients with isolated vestibular syndrome

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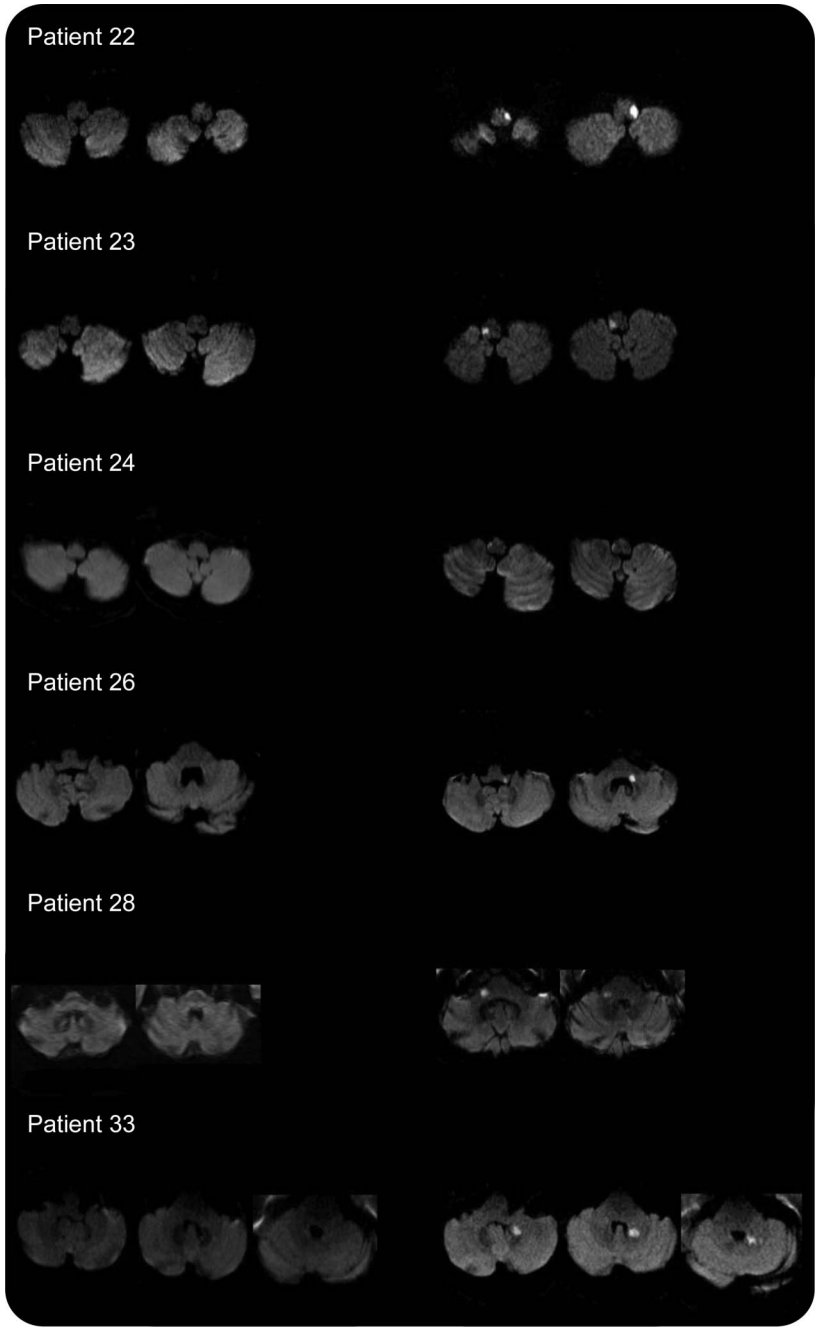
Figure 2 Initial (left panels) and follow-up diffusion-weighted images (right panels) in 6 patients with negative neuroimaging



Follow-up MRIs show infarctions in the caudal lateral medulla (patients 22–24), rostral dorsolateral medulla and inferior cerebellar peduncle (patient 26), middle cerebellar peduncle (patient 28), and inferior and superior cerebellar peduncles (patient 33).

(figure 2), and in the anteromedial medulla (n = 1) or pons (n = 3, 1 paramedian and 2 dorsal) in the remaining 4.

Clinical features

Of the 34 patients with isolated vestibular syndrome, 11 (32%) showed nystagmus (8 with spontaneous horizontal, 2 with gaze-evoked, and 1 with spontaneous downbeat and gaze-evoked nystagmus). Horizontal head impulse test was abnormal in only 1 patient with AICA

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Initial neuroimaging including DWI was falsely negative in 17.6% of patients with isolated vestibular syndrome during the early phase.

infarction (3%), and skew deviation was present in 2 (6%). Severe postural imbalance was found in 15 (44%) patients, and 6 (18%) reported severe craniocervical pain.

Most patients with isolated vestibular syndrome showed positive HINTS (32/34, 94.1%, table e-1). Two (patients 19 and 28) with negative HINTS exhibited severe postural imbalance (grade III).

DISCUSSION

The present study demonstrated that approximately 25% of patients with posterior circulation infarction present with isolated vestibular syndrome. The frequency of isolated vestibular syndrome was highest in the proximal territory infarctions, and the most commonly involved structure was the cerebellum (67.6%), mostly in the territory of medial PICA. These results are consistent with those of previous studies that showed a high frequency of medial PICA infarction in patients presenting with acute isolated vascular vertigo, and frequent isolated vertigo in medial PICA infarction.[7] Indeed, the dysmetria, a major sign of cerebellar dysfunction, may be minimal or absent in cerebellar infarctions involving the territory of medial PICA, especially when the infarction is not large.[7,22–24] In the cerebellum, the nodulus and ventral uvula may cause isolated vestibular syndrome when damaged.[9,25] The nodulus and ventral uvula receive afferents from the vestibular nuclei, nucleus prepositus hypoglossi, inferior olivary nucleus, and vestibular nerve.[e1–e4] In turn, they project to the vestibular nuclei and control the velocity-storage mechanism of the vestibulo-ocular reflex (VOR).[e5,e6] A recent study also showed that isolated nodular infarction mostly presents with isolated vertigo and imbalance without other neurologic deficits, mimicking acute peripheral vestibulopathy.[9] However, the nodulus and ventral uvula were affected in 34.8% of our patients with isolated vestibular syndrome due to cerebellar infarction. The flocculus and paraflocculus may be another neural structure leading to isolated vestibular syndrome. They participate in the control of smooth tracking, gaze-holding, and eye movements induced by vestibular stimulation, and experimental lesions cause gaze-evoked nystagmus, downbeat nystagmus, postsaccadic drift, and impaired smooth pursuit and cancellation of the VOR.[e7–e10] However, since the flocculus is supplied by a branch from the AICA, which also supplies the dorsolateral pons and inner ear, an infarction involving the flocculus usually accompanies other brainstem signs or hearing loss.[e10] In agreement with this explanation, about 74% of patients with isolated vestibular syndrome due to cerebellar infarction had lesions in the paraflocculus (tonsil), whereas the flocculus was affected in only 1.

In the brainstem lesions, vertigo is commonly associated with other neurologic symptoms and signs, but some patients with isolated vestibular syndrome showed a small lesion restricted to the vestibular nuclei or root entry zone of the eighth cranial nerve in the pontomedullary junction,[10–13] medulla,[14,15] pontine tegmentum,[16] midbrain,[17] and cerebellar peduncles.[18] We found that the ICP, SCP, and caudal lateral or rostral dorsolateral medulla are the common structures responsible for isolated vestibular syndrome in the posterior circulation infarction. Our 4 patients with an infarction involving the caudal lateral medulla presented with isolated imbalance. A 3D brainstem mapping study reported that isolated body lateropulsion in the caudal medullary lesions may be attributed to damage to the lateral vestibulospinal tract.[15] Other patients with infarctions involving the ICP, SCP, or rostral dorsolateral medulla developed imbalance with or without spontaneous horizontal nystagmus. The ICP carries various input and output fibers to and from the cerebellum, which are mainly concerned

Supplemental Data
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with integrating the proprioceptive sensory inputs with the vestibular function such as balance.[e11] Proprioceptive information from the body is carried to the cerebellum via the posterior spinocerebellar tract in the ICP.[e11] The vestibulocerebellum also receives mossy fiber inputs from the vestibular nuclei and nerve, and projects efferent fibers to the vestibular nuclei via the ICP.[e11] Thus, an infarction involving the ICP may result in imbalance with vertigo and nystagmus. However, experimental sectioning of the ICP in monkeys produced only a fall towards the side of the lesion without spontaneous nystagmus.[e12] The SCP is a primary output route of the cerebellum with the fibers carrying information to the midbrain.[e11] It also contains the fibers carrying information from the spinal cord to the cerebellum (the ventral spinocerebellar tract).[e11] Experimental lesions of the SCP usually induce a mild ipsilateral appendicular ataxia, but a severe ipsilateral body deviation also occurred in some cases.[e13] Of interest, our 2 patients with infarctions involving the SCP showed profound imbalance and spontaneous nystagmus, which reflects that the SCP may contain the fibers to control body posture from the spinal cord, and the projections from the vestibulocerebellum to the vestibular nucleus, which are responsible for the vestibulo-ocular reflex. Concurrent perfusion defects in other areas of the SCA territory may be an alternative explanation for vertigo and nystagmus in these patients. In the rostral dorsolateral medulla, isolated vestibular syndrome may occur due to an involvement of the most superior portion of the vestibular nuclei or ICP.[14]

Most of our patients (94.1%) with isolated vestibular syndrome exhibited positive HINTS. In contrast, initial neuroimaging including DWI was falsely negative in 17.6% of patients with isolated vestibular syndrome during the early phase. These results are in accord with earlier studies that demonstrated that a 3-step bedside examination for HINTS is more sensitive in detecting stroke than MRIs with DWI during the first 24–48 hours after symptom onset, while maintaining high specificity.[19] Therefore, we should decide whether we perform neuroimaging in acute vestibular syndrome on the basis of bedside examination. MRI scans are difficult to come by in some centers, and overdiagnosis of vascular vertigo would give rise to unnecessary costly workups. Also, we have to consider repeat scan in cases with unexplained or persistent signs suggestive of posterior circulation strokes on serial examination, since the first MRI scan may be negative.[19,e14,e15]

False-negative DWI results may be caused by short time interval from the symptom onset to MRI scan, since the signal-to-noise ratio could be insufficient in the first few hours after symptom onset.[19,e14] Previous studies showed that false-negative neuroimaging occurred more often when neuroimaging was obtained within 24 hours after symptom onset.[19,e14] However, our 3 patients who received neuroimaging after 24 hours from the symptom onset had initially negative results. The location or size of the lesions can explain false-negative DWI results. The lesions could be too small for the resolution of the DWI echoplanar sequence, and false-negative neuroimaging frequently occurred in cases of stroke in the posterior (19%) circulation involving the brainstem, as in our patients.[e14,e15] Magnetic susceptibility artifacts occurring in echoplanar imaging may be another possibility, which may lead to brainstem distortions that could blur image analysis.[e14] The assumption is supported by the high rate of brainstem lesions missed by DWI in the present and earlier studies.[e14,e15]

Notably, one of our patients with AICA territory infarction initially developed isolated vertigo with negative HINTS, mimicking unilateral peripheral vestibulopathy. Since the AICA supplies the inner ear, the signs of peripheral vestibulopathy may overshadow the central signs and HINTS may be negative in AICA territory infarctions. Indeed, about 5% of patients with AICA territory ischemic strokes presented with acute prolonged vertigo and canal paresis without hearing loss, mimicking peripheral acute vestibular syndrome.[e16] A recent study also showed negative HINTS in 5 of 17 patients (29.4%) with AICA infarction.[e17]

Since this study was based on the data from a single referral stroke center, a selection bias should be considered in interpreting the results. Indeed, compared to earlier studies,[2,3] the proportion of proximal infarction (34.1%) and the frequency of PICA territory infarctions (84%) in isolated cerebellar infarctions were higher, which may have increased the frequency of isolated vestibular

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syndrome and overrated the role of the structures located in the proximal territory. Furthermore, since we did not image all the patients with isolated vestibular syndrome, some patients with isolated vascular vertigo may have not been included. However, in view of the excellent diagnostic accuracy of bedside examinations for strokes in prior studies,[19,20] the proportion of such patients may be ignorable.

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DISCLOSURES

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