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Significance of Magnetic Resonance Imaging (MRI) T2 Hyperintense Endo-Vessels Sign in Progressive Posterior Circulation Infarction

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEFG Jialiang Xu
BCDE Xiaohong Chen
BDE Muhui Lin

Seventh Department of Neurology, Liaoning Province People's Hospital, Shenyang, Liaoning, P.R. China

Corresponding Author: Jialiang Xu, e-mail: batistol@126.com
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Background: MRI FLAIR hyperintense vessels sign (FHVs) is a special imaging marker that plays a key role in acute infarction imaging and diagnosis. However, FHVs have not been studied in the context of progressive posterior circulation infarction (PPCI), and little is known about the association of hyperintense endo-vessels sign (HEVs) on transverse section MRI with infarction. Thus, our objective here was to investigate the clinical significance of transverse MRI T2 HEVs in patients with PPCI.

Material/Methods: In this retrospective, case-control study, we enrolled 100 consecutive posterior circulation infarction patients. All the patients underwent head MRI examinations on the onset day and the seventh day after admission. Neurologic deficits of the patients were assessed by the National Institutes of Health Stroke Scale (NIHSS) scores upon admission and after 7 days. Infarction volume on DWI was compared.

Results: HEVs were detected in 25 of 37 patients in the PPCI group (67.6%) and 22 of 63 patients in the NPPCI group (34.9%). Logistic regression analysis showed that the proportion of HEVs in the PPCI group was higher than in the NPPCI group (P=0.007). Among all the patients, HEVs were detected in 15 of 18 patients (83.3%) with occlusion of the vertebral artery or basilar artery, and 17 of 23 (73.9%) showed severe stenosis. The proportion of vertebrobasilar artery occlusions in the PPCI group was higher than in the NPPCI group (P<0.05). MRI DWI showed that 20 patients had cerebellum infarction among 23 vertebral artery HEVs patients, and 14 patients had brainstem infarction among 15 basilar artery HEVs patients. All of the 9 vertebral and basilar artery HEVs patients had brainstem infarction. The increase in NIHSS scores from baseline to 7 days was significantly greater in patients with HEVs than in patients without HEVs in the PPCI group (P=0.002). The expansion of the infarction size from baseline to 7 days was significantly larger in patients with HEVs than in patients without HEVs in the PPCI group (P=0.037).

Conclusions: HEVs are frequently detected in patients with vertebrobasilar artery territory infarction, and they can be considered as a special imaging marker for vertebral artery and basilar artery occlusion and severe stenosis. HEVs can indicate whether or not posterior circulation infarction progresses and they may be an independent risk factor of PPCI.

MeSH Keywords: Blood Vessels • Infarction, Posterior Cerebral Artery • Magnetic Resonance Imaging

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Background

About 25% of infarctions occur in the vertebrobasilar artery system and are called posterior circulation infarctions. They are more dangerous if they involve the respiratory or circulatory centers and the brain stem reticular formation. Early detection, diagnosis, and treatment are crucial [1]. However, current clinical and imaging tests are not effective in predicting the progression and prognosis of posterior circulation infarction.

Abnormal cerebral artery hyperintensity of acute infarction patients on magnetic resonance imaging (MRI) fluid-attenuated inversion recovery (FLAIR) was first found by Cosnard in 1999, and was named the FLAIR hyperintense vessels sign (FHVs) [2]. At present, imaging evaluation of acute infarction and progression by MRI FHVs has been a research focus. The prevalence of FHVs varies according to the arterial status; moreover, FHVs are detected more frequently in patients with middle cerebral artery (MCA) occlusion. In most studies, FHVs are reported within the first day after stroke onset, but they can be detected up to 2 weeks after onset. Little is known about the clinical meaning of FHVs for posterior circulation infarction, especially for PPCI, and there have been few related studies. Studies on posterior circulation infarction FHVs have focused on the abnormality of vertebrobasilar artery shapes on MRI rather than on abnormal endo-vessels signals on MRI transverse section. Therefore, we studied acute PPCI patients to explore the association of vertebrobasilar artery HEVs with PPCI, and to explore the possible formation mechanism and clinical significance of PPCI HEVs in depth [3,4].

Material and Methods

Patients

We retrospectively screened consecutive acute posterior circulation infarction patients in Liaoning Province People's Hospital from January 2010 to October 2016. All of the patients met the criteria of the 2014 edition of the Guidelines for the Diagnosis and Treatment of Acute Ischemic Stroke in China. The patients were divided into a PPCI group and a non-progression posterior circulation infarction (NPPCI) group according to whether the infarction progressed after admission. Demographic data, risk factors of infarction, and imaging data of all patients were collected. Neurologic deficit was assessed with the NIHSS scores after admission and 7 days later. Our Institutional Review Board approved this retrospective study.

Inclusion and exclusion criteria

All the subjects had to meet the following criteria: (1) All of the patients in this study had restricted diffusion in the vertebrobasilar artery territory (cerebellum, brain stem, occipital lobe, and thalamus) on diffusion-weighted image (DWI); (2) First onset, the course of disease was less than 7 days. Exclusion criteria were: (1) Multiple infarctions other than in vertebrobasilar artery territories, or lacunar infarction; (2) Image artifacts obstructed observation; (3) Hemorrhagic cerebral vascular disease or transient ischemic attack.

According to the Scandinavian Stroke Scale (SSS), the patients in the PPCI group still had to meet the following criteria at the same time: After the third day of onset, the scores of awareness, eye movement, and physical activity compared to baseline (within 24 h of onset) decreased by 2 points with or without language score decreasing by 3 points or more. The remaining patients with posterior circulation infarction were enrolled in the NPPCI group [5].

Protocols and image analysis

All the subjects underwent advanced head MRI imaging, including DWI, T1-weighted image (T1WI), T2-weighted image (T2WI), FLAIR, and magnetic resonance angiography (MRA) at the onset day and the seventh day after admission. Head MRI examinations were performed utilizing a SIEMENS 3.0T superconducting magnetic resonance machine. MRI scanning parameters were as follows: slice thickness=5 mm, inter-slice gap=1 mm, T1WI (TR/TE=1750/25 ms), T2WI (TR/TE=8611/92 ms), FLAIR (TR/TE=8400/147 ms), DWI (TR/TE=9000/89 ms, b value=1000 s/mm^2) and 3-dimensional time of flight (3D-TOF)-MRA (TR/TE=21/2.4 ms). The images were reviewed by 2 experienced radiologists and neurologists to determine the presence of HEVs without knowing the angiographic findings [6].

HEVs were defined by endo-vertebrobasilar artery flow signals that were obviously slowed or interrupted on T2, and flow void phenomenon disappeared after being partly or completely filled by hyperintense signals (observed in transverse slices) (Figure 1). MRA findings were classified into 4 categories according to the severity of stenosis: occlusion, significant stenosis (≥50%), mild stenosis (<50%), and normal. Posterior circulation infarction was defined as brainstem, occipital lobe, thalamus, and cerebellum hyperintense lesions on DWI and corresponding hypointense lesions on apparent diffusion coefficient (ADC) maps. Infarction volume was measured in PPCI and NPPCI patients using semi-automated computerized software (Neusoft; China) [7–9]. The imaging changes are illustrated in Figures 2 and 3.

Statistical analysis

All data were processed using the SPSS 20.0 statistical software package for Windows. Normally distributed numerical data are expressed as mean differences ± standard deviation ($\bar{x}\pm s$), which was processed by using the paired *t* test. The measurement data

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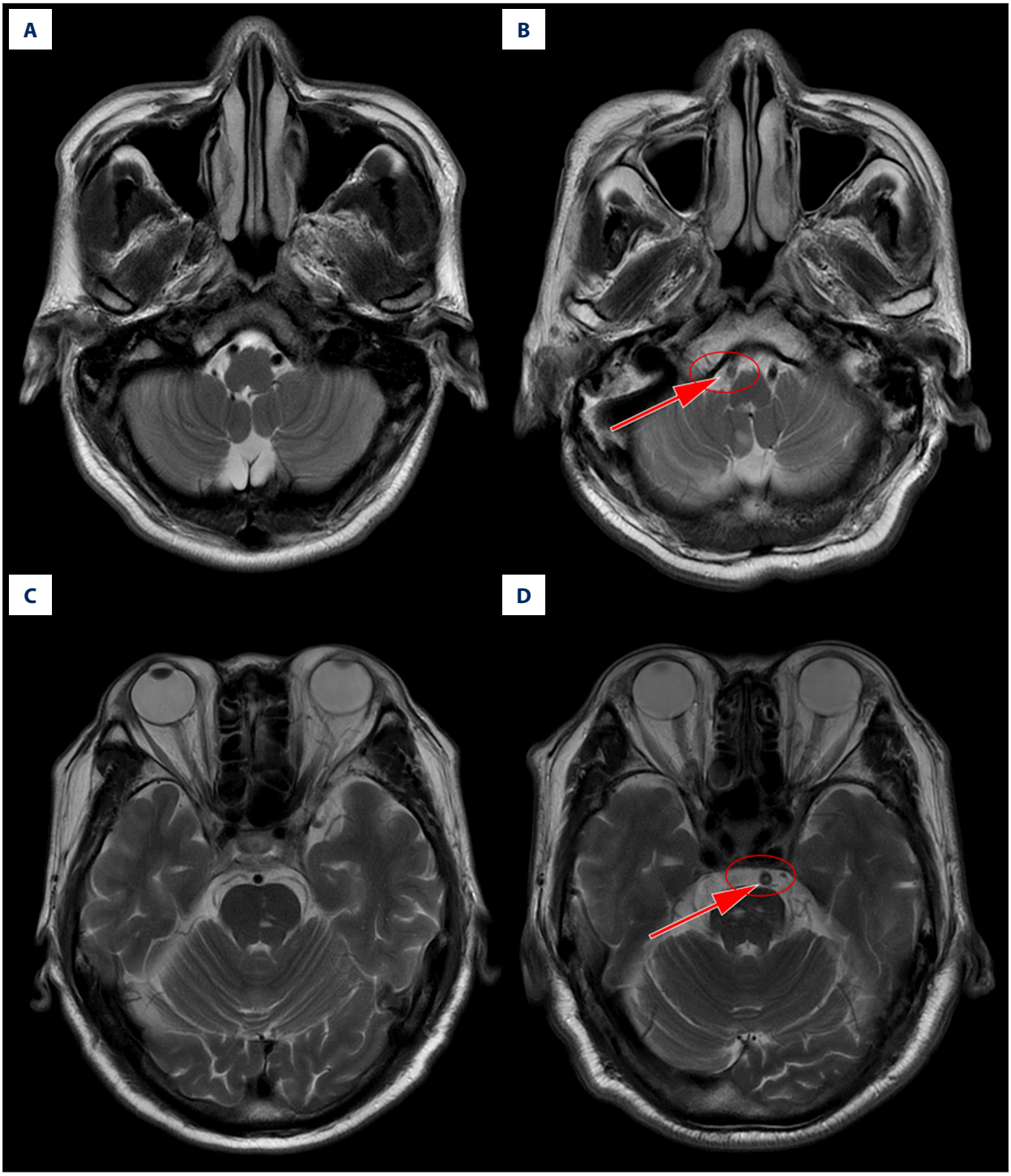

Figure 1. Vertebrobasilar artery HEVs imaging. (**A, C**) Vertebral artery and basilar artery had normal lumen, and flow void phenomenon existed on MRI T2. (**B, D**) Flow void phenomenon of vertebral artery and basilar artery disappeared and was partly filled by HEVs on MRI T2, suggesting that the endo-vertebrobasilar artery thrombosis had recently developed (arrow).

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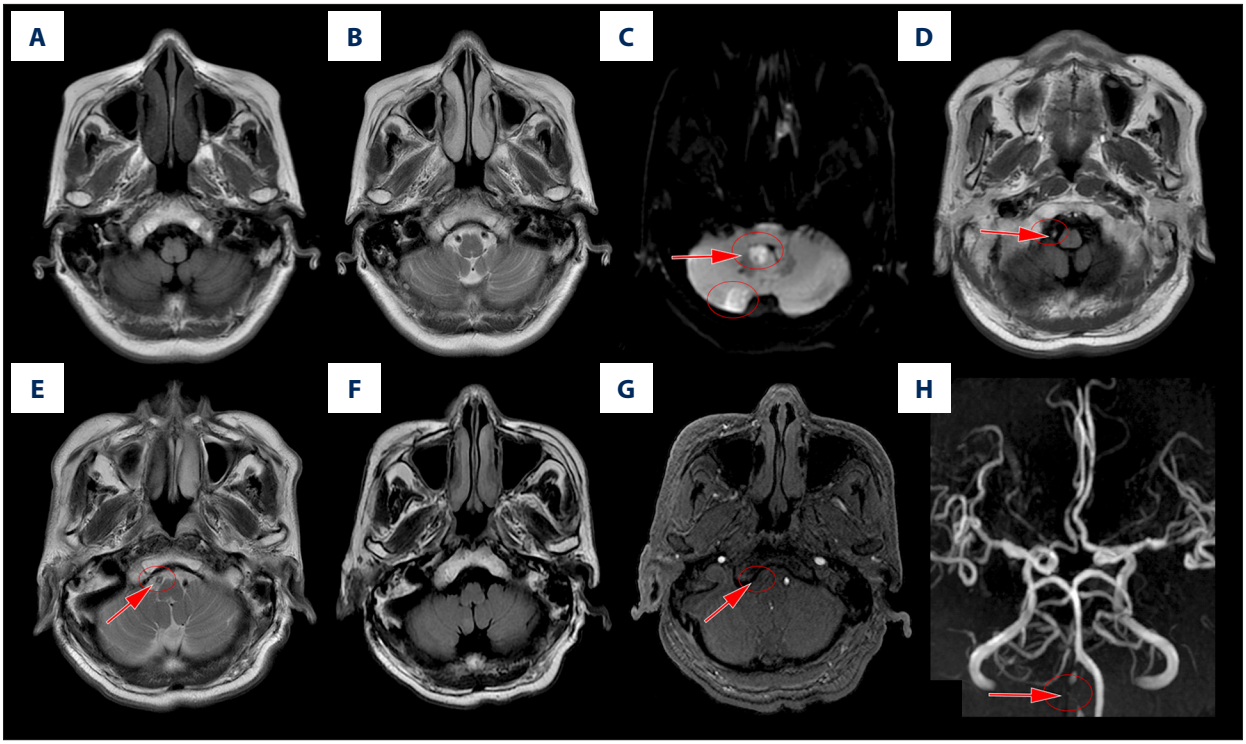

Figure 2. Example of HEVs in a PPCI patient who had the vertebral artery occlusion and the corresponding multiple cerebellum infarctions. This patient was hospitalized with dizziness and unstable walking. He suddenly began to progress on the seventh day of admission with the symptoms of dizziness, diplopia, and weakness of the right upper and lower limb. (**A, B**) MRI showed that the right vertebral artery had normal lumen, and flow void phenomenon existed on MRI T1 and T2 on the first day of admission. (**C**) DWI showed the right cerebellar hemisphere and tonsil infarction (arrow). (**D, E**) T1 and T2 showed that the blood flow void phenomenon of the right vertebral artery disappeared and HEVs were detected at the seventh day after admission (arrow). (**F**) FLAIR was normal. (**G**) The blood flow phenomenon of the right vertebral artery disappeared on 3D-TOF compared with the left side (arrow). (H) MRA indicated the possibility of right vertebral artery occlusion (arrow).

that did not conform to normal distribution were processed using the U Mann-Whitney rank sum test. The proportion of demographic data was processed by using the Pearson χ^2 test, and if the theoretical frequency was less than 5, the Fisher exact probability method was used. In addition, the Pearson χ^2 test was performed to analyze the proportion of HEVs and the difference in vertebrobasilar artery status between the 2 groups. The U Mann-Whitney test was used to compare differences in NIHSS scores and infarction volume difference between the PPCI group and NPPCI group. Logistic (forward: Wald) regression analysis was performed by taking PPCI and NPPCI as dependent variables. Age, smoking, hypertension, diabetes, HEVs, arrhythmia, coronary heart disease, hyperuricemia, and hyperhomocysteinemia were independent variables, with the odds ratio (OR value) and 95% confidence intervals (95%CI) being calculated. The level of statistical significance was $P<0.05$.

Results

Thirty-seven progressive posterior circulation infarction patients fulfilled PPCI inclusion criteria, with 23 male cases and 14 female cases. The NPPCI group included 63 patients during the same period, with 40 males and 23 females. Demographic characteristics are shown in Table 1. There was no significant statistical difference in sex, hypertension, hyperuricemia, cholesterol, homocysteine, arrhythmia, coronary heart disease, or smoking between the 2 groups ($P>0.05$). HEVs were detected in 25 of 37 patients in the PPCI group (67.6%) and 22 of 63 patients in the NPPCI group (34.9%). The proportions of vertebrobasilar artery T2 HEVs and diabetes in the PPCI group were significantly ($P<0.05$). higher than in the NPPCI group. Logistic regression analysis showed that the HEVs proportion of the PPCI group was higher than in the NPPCI group ($P=0.007$). Thus, our data suggest that HEVs were still associated with PPCI after adjustment for the other risk factors, and vertebrobasilar artery T2 HEVs might be independent risk factor of PPCI (Table 2).

Eighteen patients had artery occlusion in all the patients of the 2 groups (vertebral artery 12, basilar artery 6), and 23 patients had severe stenosis (vertebral artery 17, basilar artery 6). Twenty-five patients had mild stenosis (vertebral artery 16, basilar artery 9), and the remaining 34 patients were normal.

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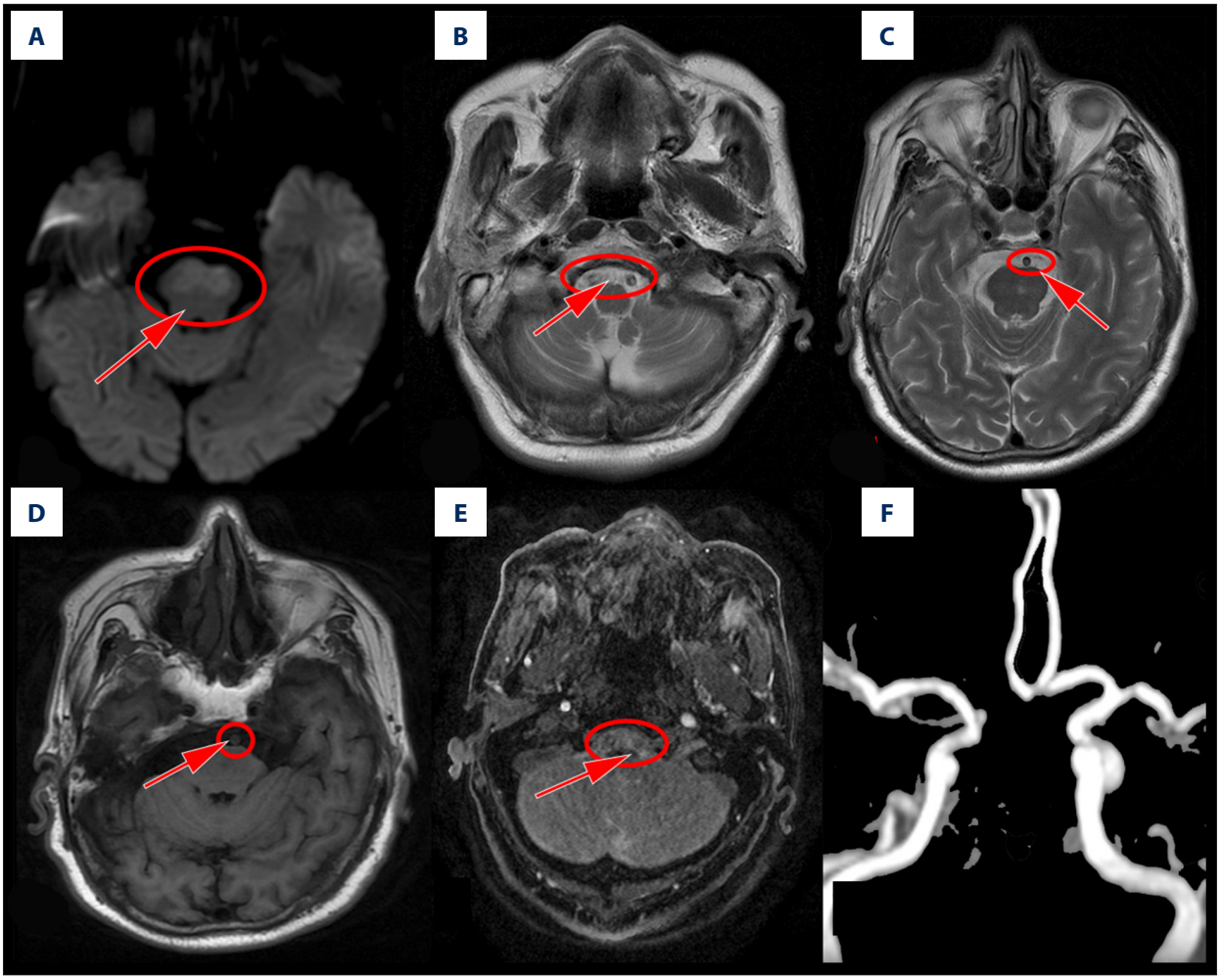

Figure 3. Example of HEVs in a PPCI patient who had the basilar artery occlusion and the corresponding bilateral middle brain cerebral peduncles infarctions. This patient was hospitalized with lack of consciousness. (**A**) DWI showed that the acute middle brain infarctions (arrow). (**B, C**) MRI T2 showed that the blood flow void phenomenon of the bilateral vertebral artery and the basilar artery disappeared and HEVs were detected (arrow). (**D, E**) MRI T1 and 3D-TOF also showed that the basilar artery flow void phenomenon disappeared (arrow). (**F**) MRA indicated the possibility of bilateral vertebra artery and basilar artery occlusion.

Among the patients, HEVs were detected in 15 of 18 patients (83.3%) with occlusion of vertebral artery or basilar artery, and 17 of 23 (73.9%) showed severe stenosis (Table 3).

Eleven patients had vertebrobasilar artery occlusion in the PPCI group, and the proportion was with the significantly ($P<0.05$) higher than in the NPPCI group (Table 4). Seven days after onset, MRI DWI showed that 20 patients had cerebellum infarction among 23 vertebral artery HEVs patients, and 14 patients had brainstem infarction among 15 basilar artery HEVs patients. All of the 9 vertebral and basilar artery HEVs patients had brainstem infarction (Table 5).

There was no difference in initial NIHSS scores in the patients with HVEs and without HEVs. The increase in NIHSS scores from baseline to 7 days was significantly greater in the patients with HEVs (6.87±7.28) than in the patients without HEVs (3.28±4.96) in the PPCI group ($P=0.002$). However, it was similar in patients with HEVs and without HEVs in the NPPCI group. There was no difference in initial ischemic DWI lesion volume between the patients with HEVs and without HEVs. The expansion of the infarction size from baseline to 7 days was significantly greater in the patients with HEVs (11.65±10.87) than in the patients without HEVs (8.29±7.87) in the PPCI group ($P=0.037$), but it was similar in the patients with HEVs and without HEVs in the NPPCI group (Table 6).

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Table 1. Clinical data comparison between the two groups.

Features	PPCI (n=37)	NPPCI (n=63)	Test statistic	P value
Age	66.4±10.4	63.9±9.2	t=1.005	0.302
Hypertension	26	42	χ^2=0.139	0.709
Diabetes	14	13	χ^2=3.855	0.043*
Smoking	11	17	χ^2=0.087	0.768
Coronary heart disease	11	16	χ^2=0.222	0.637
Arrhythmia	4	8	χ^2=0.114	0.736#
Cholesterol	5.3 (4.41±5.69)	5.1 (4.22±5.37)	U=377.500	0.536
Hyperuricemia	5	7	χ^2=0.127	0.721
Homocysteine	9	16	χ^2=0.014	0.905
T2 HEVs(+)	25	22	χ^2=9.974	0.002*

Using Fisher exact probability test; * the proportion of vertebralbasilar artery T2 HEVs and diabetes in PPCI group was higher than NPPCI group with the significant difference (P<0.05).

Table 2. Logistic regression analysis.

Factor	Wald χ^2	P	OR	95%CI
T2 HEVs	11.04	0.007	13.23	9.75–17.24
Diabetes	3.938	0.047	1.632	1.005–3.768

Table 3. Relationship between HEVs and vertebrobasilar artery status.

Artery status	Total	HEVs (+)	HEVs (–)
Occlusion			
VA	12	10	2
BA	6	5	1
Severe stenosis (≥50%)			
VA	17	13	4
BA	6	4	2
Mild stenosis (<50%)			
VA	16	8	8
BA	9	5	4
No stenosis	34	2	32

VA – vertebral artery; BA – basilar artery.

Discussion

Vertebrobasilar artery territory, which is also called the posterior circulation system, is a common type of infarction in which a posterior circulation infarction often involves brainstem respiratory and circulation centers, cough reflex center, and swallowing function. Infarction progression leads to severe adverse consequences. The prognosis of PPCI is generally poor. Therefore, clinical and radiographic research on PPCI has attracted more and more scholarly attention [10,11].

As a special imaging marker of cerebral artery occlusion and stenosis, FHVs were first reported by Cosnard [2]. The main MRI imaging features are tubular or serpentine hyperintense signals of the cerebral artery compared to gray matter on MRI FLAIR. FHVs are usually detected in the subarachnoid space

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Table 4. The comparison of vertebrobasilar artery status of PPCI and NPPCI groups.

Artery status	PPCI (n=37)	NPPCI (n=63)	test statistic	P value
Occlusion (n=18)	11	7	χ^2=5.475	0.02*
Severe stenosis (≥50%) (n=23)	9	14	χ^2=0.058	0.497
Mild stenosis (<50%) (n=25)	8	17	χ^2=0.358	0.364
No stenosis (n=34)	9	25	χ^2=0.005	0.088

* The proportion of vertebrobasilar artery occlusion of PPCI group was higher than NPPCI group with the significant difference (P<0.05).

Table 5. The relationship between the HEVs region and the hyperintense region on DWI.

HEVs in artery	Brainstem	Cerebellum	Thalamus	Occipital lobe
HEVs in VA (n=23)	16	20	1	0
HEVs in BA (n=15)	14	10	3	5
HEVs in VA+BA (n=9)	9	6	2	3

VA – vertebral artery; BA – basilar artery.

Table 6. The comparison of infarction volume and clinical outcome by HEVs between PPCI and NPPCI group.

	PPCI			NPPCI		
	HEVs (+)	HEVs (–)	P value	HEVs (+)	HEVs (–)	P value
Initial NIHSS score	4.36±5.12	4.27±5.87	0.234	3.87±4.34	3.72±4.18	0.183
7-Day NIHSS score	6.87±7.28	3.28±4.96	0.002*	2.67±3.14	2.51±3.23	0.345
Initial infarction DWI volume (mL)	7.35±6.26	7.28±6.76	0.456	7.23±6.65	7.15±6.84	0.572
7-Day infarction DWI volume (mL)	11.65±10.87	8.29±7.87	0.037*	6.24±5.34	6.17±6.21	0.782

* The increasement in NIHSS scores and the expansion of the infarction size from baseline to 7 days was significantly greater in patients with HEVs than in patients without HEVs in PPCI group (P<0.05).

due to suppression of the cerebrospinal fluid (CSF) signal, causing contrast between the dark CSF and bright blood vessels. FHVs can be observed in cerebral infarction patients with arterial occlusion or severe stenosis. Recently, because FHVs are important in assessment of acute infarction progression and prognosis, they have been studied more, becoming an important infarction neuroimaging research topic [3,12–15]. Ahn found that FHVs were frequently observed in anterior circulation infarction patients, and they often occurred in the middle cerebral artery proximal M1 and M2 segments. His study showed that FHVs were time-dependent, and they were often seen during the hyperacute or progressive period of ischemic stroke [15]. Although many studies have confirmed that FLAIR and T2 FHVs are important imaging markers of infarction, their mechanisms are unclear, and different anatomical regions have different FHVs mechanisms [1,7,16]. Early studies suggested that endo-vessel thrombosis was the main cause of FHVs, and autopsy results confirmed thrombus presence in FHV-positive vessels [8,17,18]. However, more and more studies showed that to some extent, FHVs are related to hemodynamic disorders. Liebeskind found that the formation of distal FHVs were due to the collateral circulation slow flow rather than due to endo-vessel thrombosis [19]. By studying the associations of FLAIR FHVs with infarctions on DWI, Inatomi showed that FHVs appeared earlier than DWI abnormal signals, and they were better than DWI in displaying abnormal brain perfusion areas. He thought that after cerebral artery occlusion, the leptomeningeal collaterals compensatory reflux was the pathological and physiological basis of FHVs formation [20].

Most of the previous FHVs neuroimaging studies were focused on the abnormality of intracranial and extracranial arteries shapes on MRI, but there have been few studies of abnormal endo-vessels signals on MRI transverse section [21].

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In the present study, by assessing the abnormal HEVs in PPCI patients, we found that the vertebrobasilar artery HEVs were an important imaging marker of PPCI, and we also found that the vertebrobasilar arteries in patients with HEVs had the most occlusion and more severe stenosis. MRI T2 imaging revealed that the retrograde slow flow resulted in the loss of flow void phenomenon, and hyperintense signals appeared in the endo-vertebrobasilar artery, showing that the thrombosis had recently developed. Therefore, we speculated that the possible development mechanisms of vertebral-basilar artery HEVs could be the following. The vertebrobasilar arteries exhibit pulsatile and anatomic positional changes. Those arteries flow with the occlusion or severe stenosis slowed down, and were interrupted, while collateral circulation was not fully established. This motion could lead to image blurring or signal loss and could affect the small-caliber vertebrobasilar artery in particular due to its small voxel size [22]. More and more studies have confirmed the correlation between FVHs and intracranial and extracranial artery occlusion and severe stenosis [17]. The sensitivity and specificity were 65% and 85%, respectively. Azizyan reported that HEVs were associated with ipsilateral internal carotid artery and middle cerebral artery stenosis [23]. When cerebral artery stenosis rate is ≥90%, the occurrence rate of HEVs significantly increased. Through the association study between HEVs and MRA, our study also came to a similar conclusion. Our results showed that in the patients with vertebrobasilar artery occlusion or severe stenosis, HEVs occurred more frequently, and the posterior circulation infarction patients with vertebrobasilar artery HEVs progressed faster in clinical practice than those without HEVs. Therefore, as HEVs have high sensitivity, it is reasonable to speculate that HVEs could be used to evaluate the degree of stenosis of vertebrobasilar arteries in patients with posterior circulation infarction.

At present, Chinese and foreign scholars have done some clinical research on the relationship between HEVs and the prognosis of acute cerebral infarction. By studying the relationship between HEVs and prognosis of acute anterior circulation infarction, Lee concluded that the infarction patients with HEVs were more likely to progress, and the neurological function recovery was significantly worse than in those without HEVs [24]. His study results were consistent with ours, but differed in that the subjects we chose were posterior circulation infarction patients. Our results show that the NIHSS scores of the patients with HEVs were significantly increased compared with the patients without HEVs in the PPCI group, and the infarction volume in the PPCI patients with HEVs was larger than in those without HEVs. This result suggests the clinical importance of HEVs as a prognostic factor.

Conclusions

To summarize, vertebrobasilar artery HEVs as a special imaging marker are of great clinical significance in evaluating the progression of acute infarction. In addition, HEVs on T2 have a significantly higher sensitivity for predicting vertebrobasilar artery occlusion or severe stenosis, and they were more closely associated with infarction volume on DWI. Our study is limited in that the number of patients with PPCI was not sufficient to obtain statistically significant differences. Multi-center studies with larger samples of clinical research data are needed reach more accurate clinical conclusions.

Acknowledgments

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