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Original Article

Predictive power of high-resolution vessel wall magnetic resonance imaging in ischemic stroke

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Abstract: Background and objective: Intracranial atherosclerotic disease (ICAD) is a key contributor to ischemic stroke and has a high recurrence rate. This study aimed to investigate the function of high-resolution vessel wall MRI (HR-VW-MRI) and evaluate plaque characteristics in patients with ICAD. Methods: A consecutive series of patients with ICAD who underwent HR-VW-MRI were enrolled, and imaging measurements were acquired. Baseline clinical characteristics were identified. Telephone follow-up was conducted every three months. The endpoint events were the first onset or recurrence of ischemic stroke and new clinical vascular events. Patients were divided into groups with or without events according to whether the endpoint event occurred. Results: A total of 70 patients (mean age = 57.6 years old) were enrolled. The median follow-up duration was 182 days. During the follow-up, 10 patients developed ischemic stroke, experienced endpoint events, and were found with 44 plaques in the artery area. A total of 169 plaques were further found in 70 patients. There were significant differences in EI, HST1, surface features, and WA reference between the two groups ($P < 0.05$). Logistic analysis showed that grade 2 enhancements, stenosis degree ≥ 50%, HST1, and surface features were independent prognostic factors of the onset of stroke, caused by ICAD. Conclusion: This prospective study demonstrates that HR-VW-MRI can identify atherosclerotic plaques in the cerebral artery and high-risk plaques, which may contribute to the prevention of ICAD and guide clinical treatment.

Keywords: Intracranial atherosclerotic disease, ischemic stroke, plaque features, high-resolution vessel wall MRI

Introduction

Intracranial atherosclerotic disease (ICAD), a pivotal factor contributing to ischemic stroke, frequently causes death and long-term disability [1, 2]. Despite antiplatelet treatment and control of associated risk factors, the annual stroke recurrence rate of symptomatic ICAD is as high as 12.6%. Traditional methods, including digital subtraction angiography (DSA), computed tomography angiography (CTA), and magnetic resonance angiography (MRA), have been used to evaluate luminal stenosis for the diagnosis of ICAD; however, luminal narrowing is not a satisfactory predictor of plaque, owing to compensatory remodeling [3, 4].

High-resolution vessel wall magnetic resonance imaging (HR-VW-MRI) is an appropriate tool for evaluating the characteristics of the extra cranial vessel wall [5, 6] and is further used to examine intracranial vessels. It is also commonly used to evaluate the atherosclerosis [7-9]. The variable refocusing flip angle (VRFA) technique, the most widely acknowledged 3D sequence, allows for them on ochrome imaging of blood and the generation of outstanding image quality with a large monitor coverage in a short scan time [10, 11]. The VRFA can be further improved by adding a pre-pulse, like delay alternating with nutation for tailored excitation (DANTE), which can optimize cerebrospinal fluid and suppress blood flow so that flow artifacts are limited [12, 13]. Motion-sensitized driven equilibrium (MSDE) can also be added, which can suppress blood further, using flow-sensitive dephasing gradients [14]. However, the scan time usually increases with the addition of preparation pulses. Some existing improved technologies can be jointly used to image intracranial and carotid vessel walls in an isotropic resolution scan [15-17].

The application of these techniques provides more effective approaches to the exploration of the underlying mechanisms of ischemic stroke and its recurrence. Some related studies provide the results of HR-VW-MRI in intracranial artery diseases [18-20]. However, so far, there are few reports on the prognostic value of HR-VW-MRI in predicting stroke. This study evaluates the plaque characteristics in patients with ICAD using HR-VW-MRI.

Materials and methods

Patients

This study was approved by the Medical Ethics Committee of the Second Affiliated Hospital of Xinjiang Medical University (Urumqi, China) (Ethical approval no. 20180227-06). All the participants provided written informed consent. This study's participants were patients from the stroke center from August 2017 to December 2019.

The inclusion criteria were: (1) Patients with evidence of ICAD lesions that caused any degree of stenosis in a large intracranial artery and were confirmed by a prior CTA, DSA, or MRA scan; (2) Patients with more than one cardiovascular risk factor, such as history of smoking, obesity, hypertension, hyperlipidemia, and diabetes mellitus [21]; (3) Patients with no contraindications for MRI; and (4) Patients with positive attitude toward medical treatment. The exclusion criteria were: (1) Patients with over 50% stenosis in the extracranial cervical artery ipsilateral to the diseased intracranial vessels; (2) Patients with evidence of non-atherosclerotic intracranial vasculopathy (e.g., dissection, reversible cerebral vasoconstriction syndrome, vasculitis, or Moyamoya disease); (3) Patients with cardioembolism; and (4) Patients with uninterpretable MR images caused by patient motion. Clinical characteristics were also collected and used for prospective analysis.

MRI examination

A 3.0-T MRI scanner (Achieva; Philips Healthcare, Best, the Netherlands) with a 32-channel head coil was used to obtain the MRI scans. The sequences of intracranial vessels were obtained after a routine brain MRI scan (T1WI, T2WI, FLAIR, and DWI), and 3D HR-VW-MRI was also conducted. Before and after injection of contrast agents, all the patients underwent sagittal 3D T1W DANTE-VISTA sequence scans. The parameters were as follows: TR/TE: 800 ms/20 ms; TSE factor: 45 echoes; field of view: 180×163×140 mm^3; acquired resolution: 0.5×0.5×0.5 mm^3 and acquisition time: 7 min 48 s. Post contrast DANTE-VISTA sequence scan was performed five minutes after the patients were administered with gadolinium-based contrast agents (0.1 mmol/kg per patient's weight).

Image analysis

Images were analyzed by 2 independent readers who did not know each other. In case of a disagreement, a consensus was reached through discussion.

Image quality was first evaluated using a four-point scale (1, failure; 2, poor; 3, adequate and 4, excellent). Images with unclear presentation of the vascular wall structure were regarded as poor. Images with the best presentation of the vascular wall structure were regarded as excellent or adequate. Images rated as adequate or excellent were analyzed, and poor images were excluded.

The vascular territories were divided as follows: the cavernous (C3) and supra clinoid (C4) segments of the internal carotid artery (ICA), the M1 and M2 segments of the middle cerebral artery (MCA), A1 and A2 segments of the anterior cerebral artery (ACA), basilar artery (BA), V4 segment of the vertebral artery (VA), and P1 and P2 segments of the posterior cerebral artery (PCA). All plaques detected in each vessel segment were also recorded.

The vascular images were reconstructed in multiple planes along the longitudinal axis of the targeted artery and orthogonal to the targeted artery. If there was an atherosclerotic plaque on HR-VW-MRI, this was defined as an eccentric vessel wall thickening regardless of the degree of stenosis. Quantitative MRI measurements included the outer wall area (OWA), lumen area (LA), remodeling ratio (RR), area-based stenosis degree, wall area (WA: OWA-LA), and plaque burden (WA/OWA ×100%). RR is the ratio of the lesion OWA compared to the reference OWA. The reference site was defined as the normal vessel segment proximal

Table 1. Basic clinical characteristics of ICAD patients

Patient characteristic	n (%) or median (range)
Age (mean ± SD, years old)	57.6 (36~76)
Men	51 (72.9%)
Female	19 (27.1%)
Smoker	31 (44.3%)
Diabetes mellitus	24 (34.3%)
Hypertension	39 (55.7%)
Hyperlipidemia	35 (50.0%)
Stroke history	12 (17.1%)
Event group	10 (14.3%)

or distal to the maximal stenotic site. If unavailable, the contralateral artery was used. RR > 1.05 was considered positive for remodeling, 0.95 ≤ RR ≤ 1.05 as intermediate, and RR < 0.95 as negative [22]. A high signal on T1WI (HST1) was a high signal area within the plaque, and the signal intensity (SI) was over 150% of the reference [23]. The morphologic characteristics of the plaque surface were defined as regular or irregular. Plaque enhancement was divided into three levels [24]: Grade 0: ≤ the degree of enhancement of the normal intracranial vascular wall; Grade 1: between the degree of enhancement of the normal vessel wall and the degree of enhancement of the pituitary infundibulum; Grade 2: ≥ the degree of enhancement of the pituitary infundibulum. According to the SI measured on the pre- and post-contrast T1W VISTA images, the enhancement index (EI) was obtained to quantitatively evaluate the enhancement degree of the plaque. The SI of plaques (SI plaque) on T1W VISTA images matched by the pre- and post-contrast and SI of adjacent normal gray matter (SI gray matter) were evaluated at the level where intracranial arterial plaque enhancement was the most obvious. The plaque EI was determined to indicate the degree of plaque enhancement using the following equation: EI = [(SI plaque/SI gray matter on post contrast T1WI) - (SI plaque/SI gray matter on pre contrast T1WI)]/(SI plaque/SI gray matter on pre contrast T1WI) ×100%.

Follow-up

All the patients were treated according to the Chinese guidelines for the treatment of ischemic stroke [25]. Telephone follow-up was conducted every three months after discharge to inquire about current medication, signs of cerebrovascular events, risk factors, and the first onset and recurrence of ischemic events. Primary outcome events included the first onset of ischemic stroke in patients who received primary prevention and recurrent ischemic stroke in patients who received secondary prevention. Secondary endpoints were new clinical vascular events, including intracerebral hemorrhage, myocardial infarction, and vascular death. When neurological impairment was worse than before or new neurological symptoms or signs indicated cerebral infarction, patients were told to visit the doctor in time and undergo cerebral MRI examination, such as DWI, to ascertain the location of the cerebral infarction. The recurrence or progression of stroke was monitored by clinical physicians who did not know the HR-VW-MRI plaque characteristics. The patients were assigned to the event group and non-event group based on the results of the head MRI.

Statistical methods

The SPSS software (version 17.0; SPSS Inc., Chicago, IL, USA) was used for data analyses. Mean ± standard deviation (SD) was used to present continuous variables; the frequency and percentage were used for categorical variables. An independent sample *t*-test was conducted to compare continuous variables; the Chi-square test was used for categorical variables. A univariate logistic regression analysis was further performed to determine the relationship between the event and risk factors. A binary logistic regression model was used to evaluate the relationships between event plaques and MRI measurements. Statistical significance was set at $P < 0.05$.

Results

ICAD patients and plaque characteristics

A total of 70 ICAD patients (51 male and 19 female) with a mean age of 57.6 (range, 36-76) years old; hypertension, 39 (55.7%); dyslipidemia, 35 (50.0%); diabetes, 24 (34.3%); smoking, 31 (44.3%) and previous stroke, 12 (17.1%) were enrolled as shown in **Table 1**. The median follow-up time and interquartile inter-

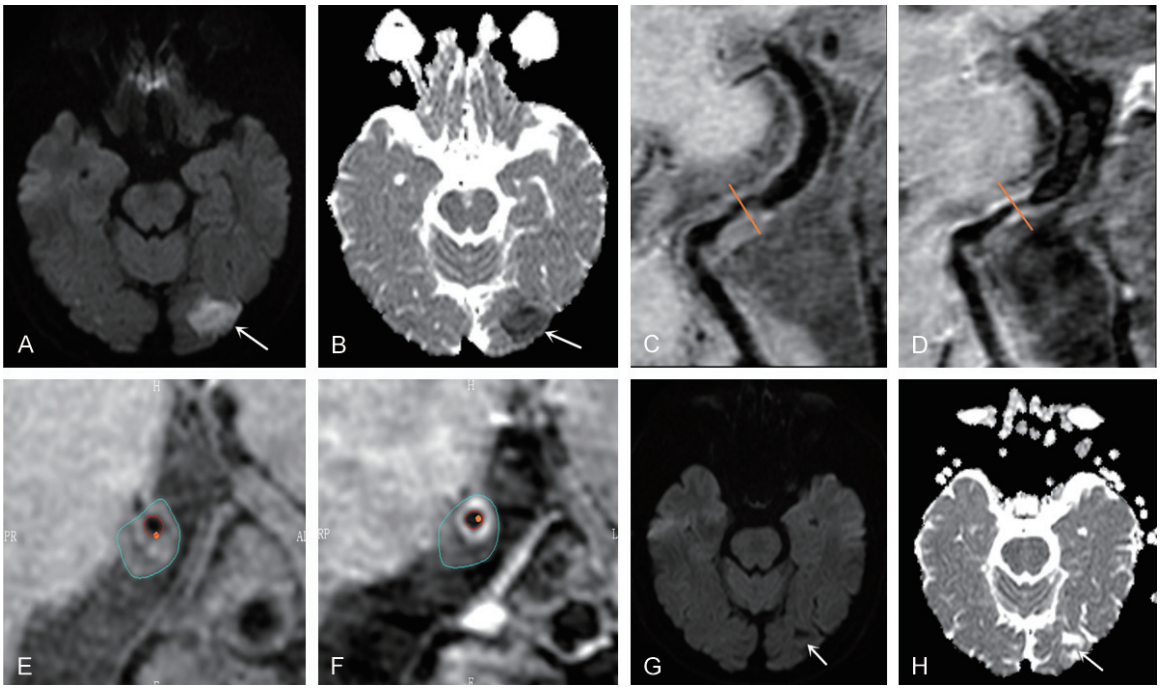

Figure 1. A 62-year-old symptomatic ICAD patient with severe stenosis on the BA. DWI detected a lesion with hyperintensity (A, white arrow) in the left occipital lobe, which showed hypointensity on ADC (B, white arrow). Long-axis images (C, D) reconstructed from pre- and post-contrast enhancement HR-VW-MRI volume acquisition revealed a focal plaque located at the BA. Short-axis images (E, F) were reconstructed from the pre- and post-contrast enhancement HR-VW-MRI orthogonal to the BA. The contour of the outer wall is in blue, and the lumen is in red. (G, H) Were images after re-examination. Hypointensity lesion (G, white arrow) was seen in the left occipital lobe on DWI and hyperintensity on ADC (H, white arrow).

Table 2. Comparison of MRI measurements of plaques between the 3 groups

	non-event group	event group	*P*
Remodeling ratio	1.05±0.33	1.06±0.26	0.911
Stenosis degree (%)	49.05±28.78	54.87±29.41	0.253
EI (%)*	49.19±42.38	69.09±42.80	0.008
HST1, n (%)*	6 (4.8)	18 (40.9)	0.001
Surface irregularity, n (%)*	7 (5.6)	18 (40.9)	0.001
Lesion site			
Lumen area (mm^2)	3.67±3.50	3.51±3.47	0.803
Wall area (mm^2)	14.58±5.95	16.96±7.47	0.061
Outer wall area (mm^2)	18.25±8.66	20.47±10.21	0.165
Plaque burden (%)	82.75±10.04	85.36±9.67	0.136
Reference site			
Lumen area (mm^2)	6.25±3.10	7.16±4.52	0.224
Wall area (mm^2)*	10.87±2.71	11.91±3.29	0.040
Outer wall area (mm^2)	17.13±5.59	19.08±7.60	0.074

EI indicates enhancement index; HST1, high signal on T1WI; *P < 0.05.

val were 182 and 155 days, respectively. During the follow-up period, 10 patients suffered ischemic stroke, of which six had recurrent ischemic stroke, while four suffered the first onset of ischemic stroke.

A total of 169 plaques were found in 70 patients, of which 68 were located in the anterior circulation and 101 in the posterior circulation, as shown in **Figure 1**. There were 44 plaques detected in the supplying vascular region of the infarcted lesions in 10 patients who suffered ischemic stroke endpoints.

Analysis of plaque characteristics between event group and non-event group

Statistically significant differences were found in EI, HST1, surface irregularity, and the WA of the reference site ($P < 0.05$) when the MRI measurement indexes were compared between the event and non-event groups. Other MRI measures showed no significant differences, as shown in **Table 2**.

Table 3. Logistic regression analyses in event and non-event plaques

	Univariate Analysis		Multivariable Analysis	
	OR (95% CI)	*P*	OR (95% CI)	*P*
HST1 (yes vs. no)	13.73 (4.96-37.95)	0.001	5.18 (1.53-17.49)	0.008
Surface features (irregular vs. regular)	11.67 (4.42-30.81)	0.001	5.97 (1.79-19.85)	0.004
Stenosis degree (≥ 50% vs. < 50%)	4.50 (2.08-9.72)	0.001	4.68 (1.83-11.98)	0.001
Enhancement grade (Grade 1 vs. Grade 0)	1.97 (0.79-4.95)	0.146	1.86 (0.64-5.41)	0.249
Enhancement grade (Grade 2 vs. Grade 0)	9.48 (3.62-24.87)	0.015	4.37 (1.33-14.32)	0.015

HST1 indicates high signal on T1WI; CI, confidence interval; OR, odds ratio.

Associations of risk factors with plaques

The univariate logistic regression analysis showed that grade 2 enhancement, HST1, surface irregularity and stenosis rate ≥ 50% were risk factors for plaques in the event group. Variables that displayed a univariate correlation with the event were applied to the multivariate logistic regression model. Multivariate logistic regression analysis showed that grade 2 enhancements, HST1, surface irregularity, and stenosis rate ≥ 50% were independently associated with the event (**Table 3**).

Discussion

HR-VW-MRI was used to analyze the prognostic factors of ischemic stroke. The results indicated that stenosis rate ≥ 50%, grade 2 enhancement, HST1 and surface irregularity were independent prognostic factors of stroke caused by ICAD.

This study found that grade 2 enhancement on HR-VW-MRI was an independent prognostic factor of stroke caused by ICAD, which was consistent with previous results [24, 26]. The stroke risk of patients with grade 2 enhancement plaques was approximately 4.37 times higher than that of patients without enhanced plaques. A previous related study reported that plaque enhancement was a risk factor for stroke recurrence after adjustment for stroke and MRA stenosis history [26]. It was speculated that most stroke recurrences occurred within one month after the initial onset of ischemic stroke, and that the atherosclerotic plaques might be vulnerable in recurrent cases, which could be accurately determined using new imaging methods. A related study investigated the association between plaque enhancement and stroke recurrence and further investigated the key imaging indicators for predicting stroke recurrence, and the results showed that plaque enhancement was commonly observed in patients with intracranial atherosclerotic stroke. The baseline plaque enhancement data were related to both the recent event and the recurrence of ischemic stroke. Combined with infarction type and collateral circulation status, plaque enhancement could better predict the risk of stroke recurrence [24]. These studies also showed that the use of gadolinium contrast agents could ascertain plaque enhancement, which serves as a marker for neovascularization, inflammation, and plaque instability. The degree of plaque enhancement reflected inflammation levels, as enhanced permeability of endothelial cells and neovascularization were independently related to the recent onset of cerebral ischemia. Plaque enhancement is an important sign for analysis during MRI, as it can be determined and evaluated without complex image post-processing [20, 27].

The degree of stenosis of the intracranial artery is a significant indicator of stroke recurrence. One related study used HR-VW-MRI to examine the features of patients with intracranial atherosclerotic plaque enhancement and the association between the enhancement of intracranial vascular plaque and the recent onset of ischemia [28]. The results of this study, as with the others [29, 30], suggested that atherosclerotic plaque enhancement could result in worse lumen stenosis and may be a sign of plaque instability. This study also indicated that the stenosis of the lumen had an independent relationship with the enhancement of plaque, which was an independent factor that indicated a recent onset of ischemia. From a large sample size research, compared with patients without enhanced plaques, more patients with enhanced plaques had stenosis > 50% on conventional MRA [26]. Further, the positive rate of

MRA in the group with enhancement was significantly higher than that in the group without enhancement. Moreover, clinical data and plaque features were analyzed in patients with and without plaque enhancement. It was suggested that stenosis > 50% might lead to stroke, as mentioned in related literature [28].

The plaque with IPH which is another related factor tended to have a high signal area, which was considered an embolism caused by intracavitary thrombosis [31]. Multivariate analysis demonstrated that IPH and irregularity of the plaque surface were independently associated with arterial-to-artery infarction. Studies on coronary arteries also indicated that IPH was associated with plaque instability [32]. IPH is caused by the rupture of microvessels with thin walls and intra plaque vessels. Some related studies demonstrated that paraluminal bleeding/thrombosis suggesting rupture or erosion, might contribute to the recurrence of thrombosis. A related pathological study confirmed that HR-VW-MRI could distinguish IPH from paraluminal bleeding/thrombosis in carotid atherosclerotic diseases [33]. IPH could be employed as a new imaging predictor for stroke recurrence and as a marker for the individualized evaluation of anticoagulant therapy.

Plaque surface irregularity was also observed in the event group in this study. Previous related studies show that an embolism might detach when there are surface irregularities (> 2 mm) [34]. Patients with arterial-to-artery infarction have been found to have a higher incidence of irregularity of the plaque surface and IPH than those without arterial-to-artery infarction. The coexistence of these features might result from plaque protrusions or fragmentation, which could increase the onset of thrombosis on the surface.

The present study has some limitations. First, the ICAD lesions identified on HR-VW-MRI were not validated by histopathology. Second, the study's sample size was small, and the results might not be sufficiently robust. Further prospective research with a larger sample size and a longer follow-up period should be performed. In conclusion, although patients with cardiac embolism were excluded, in situ stenosis and exfoliated embolism could not be accurately distinguished.

Conclusion

HR-VW-MRI can provide more information and is suitable for the diagnosis of ICAD. This might provide new insights into intracranial vascular pathology. Based on HR-VW-MRI and key clinical data, it can evaluate the progress or pathogenic risk of ICAD, contribute to individualized treatment strategies, optimize the effect of primary and secondary prevention and reduce the incidence and recurrence rate of ischemic stroke.

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Disclosure of conflict of interest

None.

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