

# Vessel Wall MRI of Chronic Internal Carotid Artery Occlusion: Imaging Findings Associated with Recurrent Stroke

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## Research Article

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# Abstract

## Purpose

The purpose of this study was to identify the vessel wall magnetic resonance imaging (VWI) features predictive of stroke recurrence in patients with chronic internal carotid artery occlusion (CICAO).

## Materials and methods

Data of 188 patients with CICAO examined by HR-MRI from October 2018 to September 2025 were retrospectively analyzed. The 188 patients were divided into three groups: group 1 (acute recurrent stroke), group 2 (acute first stroke), and group 3 (chronic stroke). Clinical characteristics and HR-VWI vessel wall features were compared between the three groups.

## Results

Diabetes prevalence, elevated HbA1c, left-sided lesion, and hyperintensity in distal occluded vessel lumen were significantly more common in group 1 than in group 2 or group 3 (diabetes,  $P_{\text{all}} = 0.029$ ; HbA1c,  $P_{\text{group 1 vs. group 3}} = 0.01$ ; left-sided lesion,  $P_{\text{group 1 vs. group 2}} = 0.012$ ,  $P_{\text{group 1 vs. group 3}} = 0.003$ ; hyperintensity in distal occluded vessel lumen,  $P_{\text{group 1 vs. group 2}} = 0.01$ ;  $P_{\text{group 1 vs. group 3}} < 0.001$ ). Ipsilateral posterior communicating (Pcom) score  $\geq 4$  points was significantly less common in groups 1 and 2 than in group 3 (37.3% vs. 14.3% vs. 55.1%;  $P_{\text{group 1 vs. group 2}} = 0.014$ ,  $P_{\text{group 2 vs. group 3}} < 0.001$ ,  $P_{\text{group 1 vs. group 3}} = 0.027$ ), and was higher in group 1 than in group 2. FLAIR vascular hyperintensity (FVH) was significantly more common in groups 1 and 2 than in group 3 (60% vs. 61.33% vs. 24%;  $P_{\text{group 1 vs. group 3}} < 0.001$ ,  $P_{\text{group 2 vs. group 3}} < 0.001$ ). In multivariate analysis, the independent predictors of stroke recurrence were left-sided lesion (OR, 2.54; 95% CI: 1.05–6.14,  $P = 0.04$ ), distal occluded vessel lumen hyperintensity (OR, 2.86; 95% CI: 1.16–7.06;  $P = 0.02$ ), and ipsilateral Pcom score  $< 4$  (OR, 3.48; 95% CI: 1.15–10.55,  $P = 0.03$ ).

## Conclusion

Patients with left-sided lesion, distal occluded vessel lumen hyperintensity, and poor primary collateral flow have higher risk of CICAO stroke recurrence.

## Introduction

Chronic internal carotid artery occlusion (CICAO) accounts for 6.5% of all ischemic strokes (1). In patients with symptomatic CICAO, the recurrence rate is 10%–18% per year and the mortality rate approximately 9.5% (2–4). Repeated ischemic strokes may result in cognitive decline (decreased

psychomotor speed, executive function, and working memory), disability, and death (5, 6), and hence prevention of stroke recurrence is extremely important.

Stroke recurrence is associated with vascular risk factors such as hemodynamic disorder, thromboembolism, occlusion pattern, and collateral reserve status (7, 8). Evaluation of vascular risk factors requires detailed knowledge of the anatomy of the occluded vessel and the secondary changes in the distal vessels. Intraluminal imaging techniques (ultrasound, computed tomography angiography, magnetic resonance angiography, and digital subtraction angiography) (9–12) are useful for evaluating the condition of the occluded stump, occlusion length, distal reflux, and collateral circulation, but provide limited insight into the pathological features of the vessel wall and the lumen of the occluded segment.

VWI is an effective and repeatable technique for assessment of intracranial and extracranial vascular disease, allowing direct visualization of the vessel wall and occluded lumen, the composition of the thrombus or plaque, the length of the occluded segment, the collateral circulation, and the path of the occluded vessel (11, 12). However, the HR-MRI characteristics of occluded vessels and the secondary changes in distal vessels in patients with CICA0 and ischemic stroke recurrence have not been sufficiently clarified. The purpose of this study was to identify the VWI features associated with stroke recurrence in patients with CICA0. The findings of this study could help optimize clinical management of strokes and prevent future recurrences.

## Methods

### Study design and population

The data of 412 patients with internal carotid artery occlusion treated at Shaanxi Provincial People's Hospital from October 2018 to September 2025 were extracted from the picture archiving and communication systems database for retrospective analysis. Patients were eligible for inclusion if they had (13): 1) internal carotid artery occlusion diagnosed by ultrasound, computed tomography angiography (CTA), magnetic resonance angiography (MRA), or contrast enhanced magnetic resonance angiography (CEMRA); 2) chronic internal carotid artery occlusion was diagnosed for more than 4 weeks, confirmed by digital subtraction angiography (DSA); and 3) undergone both routine magnetic resonance imaging (MRI) and head and neck vascular VWI. The exclusion criteria were 1) ipsilateral middle or anterior cerebral artery stenosis > 50% or unstable plaque; 2) contralateral internal carotid artery stenosis > 50%; 3) image quality inadequate for evaluation; 4) nonatherosclerotic occlusion (e.g., due to dissection or moyamoya disease, with evidence of cardiac embolism); or 5) no ischemic stroke in the territory of internal carotid artery occlusion. The patients were separated into three groups: group 1 comprised patients with acute recurrent stroke diagnosed by imaging (VWI, routine MRI and DWI ) within 4 weeks of the stroke recurrence after presentation; group 2 comprised patients with first acute stroke diagnosed by imaging within 4 weeks of stroke onset after presentation; and group 3 comprised patients with chronic stroke (i.e., imaging performed > 12 weeks of stroke after presentation). Data were

collected on age, sex, hypertension, diabetes, HbA1c, dyslipidemia, coronary artery disease, smoking as current smoking, current drinking, uric acid, homocysteine, aspirin use, statin use, interval time examination and MRI features.

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Shaanxi Provincial People's Hospital (NO.2018K-S042), and all patients have signed the informed consent form.

## MRI

MRI was performed using a Philips 3.0T Ingenia MRI scanner, equipped with an 8-channel cervical vascular wall coil and a 32-channel head coil (Philips Healthcare, The Netherlands). The head scanning sequences included T1 weighted imaging (T1WI), T2 weighted imaging (T2WI), fluid attenuated inversion recovery (FLAIR), and diffusion weighted imaging (DWI); the parameters were as follows: field of view (FOV) =  $230 \times 180 \text{ mm}^2$ , acquisition matrix =  $480 \times 480$ , slice thickness = 6 mm, and slice gap = 1 mm. HR-VWI sequences included three dimensional T1WI volume isotropic turbo spin-echo acquisition (3D-T1WI-VISTA), contrast-enhanced 3D-T1WI-VISTA, black blood-T1WI (BB-T1WI), and black blood-T2WI (BB-T2WI); the scanning parameters were as follows: FOV =  $200 \times 200 \text{ mm}^2$ , repetition time/echo time (TR/TE) = 1000/16 ms, acquisition matrix =  $400 \times 400$ , voxel size =  $0.5 \times 0.5 \times 0.5 \text{ mm}^3$ , number of signals averaged (NSA) = 1, acceleration factor = 2. The parameters for BB-T1WI were: FOV =  $140 \times 101 \text{ mm}^2$ , TR / TE = 477/13 ms, acquisition matrix =  $312 \times 216$ , voxel size =  $0.45 \times 0.45 \times 2 \text{ mm}^3$ , NSA = 2. The parameters for BB-T2WI were: FOV =  $140 \times 101 \text{ mm}^2$ , TR / TE = 3000/90 ms, acquisition matrix =  $312 \times 216$ , voxel size =  $0.45 \times 0.45 \times 2 \text{ mm}^3$ , NSA = 2. Enhanced scanning was performed after intravenous administration of gadoterate meglumine 0.1 mmol/kg over 5 minutes. Total scanning time was ~ 35 minutes.

## Imaging Analysis

VWI raw data were imported into the Plaque View post-processing software (VP Diagnostics). Two neuroradiologists, each with 5 years of experience in HR-VWI and blinded to clinical information, independently analyzed the occluded internal carotid artery stump, proximal plaque composition, distal occluded vessel lumen signal, distal reflux, collateral circulation, and FLAIR vascular hyperintensity (FVH). Disagreements were resolved by discussion. The collateral circulation was divided into primary (anterior or posterior communicating artery) and secondary (ophthalmic and meningeal arteries), and evaluated on 3D-T1WI-VISTA. Primary collateral and ophthalmic arteries were graded as follows: 1, absent; 2, probably present; 3, wire sample; 4, definitely present; or 5, strong; grades 4–5 were considered to indicate good collateral flow. Meningeal arteries were graded on a scale of 0 to 3, with grades 2–3 indicating good collateral flow (14–16). Distal occluded lumen hyperintensity at the end of the distal lumen was defined as signal intensity of the occluded lumen higher than that of the

sternocleidomastoid signal at the same level on 3D-T1WI-VISTA (17). The stump was defined as the proximal end of the occluded segment of the common carotid artery bifurcation to the ICA with contrast medium filling. VWI criteria were used to identify lipid-rich necrotic core, calcifications, and hemorrhages within the plaque (18). FVH was defined as striated, punctate, or tubular high signal seen in the subarachnoid space on FLAIR in the territory of intracranial macrovascular occlusion/stenosis (19).

## Statistical Analysis

SPSS 26.0 (IBM Corp., Armonk, NY, USA) was used for data analysis. The normality of distribution of continuous variables was first tested. Normally distributed variables were summarized as means  $\pm$  standard deviations and non-normally distributed variables as medians (with interquartile ranges [IQR]); the Mann–Whitney *U* test for comparison between groups. Categorical variables were expressed as counts or percentages, and the chi-square test or Fisher exact test was used for comparison between groups. Bonferroni correction was applied to all of the above for two-way comparisons between groups. Interobserver agreement was quantified using the kappa coefficient. Variables significantly associated (at  $P < 0.1$ ) with stroke recurrence in univariate analysis were entered into backward multivariate regression analysis to identify the independent related indicators of stroke recurrence. Receiver operating characteristic curves (ROC) were drawn for each independent predictor, and the area under the curve (AUC) was calculated to assess predictive ability.  $P < 0.05$  was considered statistically significant.

## Results

### Patient Demographics

Figure 1 shows the patient selection process. The 188 patients were divided into three groups. Group 1 comprised 75/188 (39.9%) patients with mean age of  $64.45 \pm 11.98$  years, interval stroke recurrence and from stroke onset to imaging examination ( $14.7 \pm 6.6$ , months); group 2 comprised 35/188 (18.6%) patients with mean age of  $65.54 \pm 12.04$  years; and group 3 comprised 78/188 (41.5%) patients with mean age of  $69.56 \pm 11.71$  years, interval from stroke onset to imaging examination ( $16.1 \pm 4.2$ , months). Mean age was significantly higher in group 3 than in group 1 ( $69.56 \pm 11.71$  years vs.  $64.45 \pm 11.98$  years,  $P = 0.024$ ). Diabetes prevalence was significantly higher in group 1 than in the other two groups ( $P_{\text{group 1 vs. group 2}} = 0.039$ ,  $P_{\text{group 1 vs. group 3}} = 0.024$ ). HbA1c was significantly higher in group 1 than in group 3 ( $6.57$  [IQR, 6.14–7.0] vs.  $5.68$  [IQR, 5.43–5.92],  $P = 0.01$ ). Other variables were comparable between the three groups (Table 1).

Table 1  
Comparison of clinical characteristics of CICA0 groups

| Age, yr                    | Group 1<br>(acute<br>recurrent<br>stroke, n<br>= 75) | Group 2<br>(acute first<br>stroke, n =<br>35) | Group 3<br>(chronic<br>stroke,<br>n = 78) | <i>P</i> <sub>all</sub> | <i>P</i> <sub>group<br/>1 vs.<br/>group 2</sub> | <i>P</i> <sub>group<br/>1 vs.<br/>group 3</sub> | <i>P</i> <sub>group<br/>2 vs.<br/>group 3</sub> |
|----------------------------|------------------------------------------------------|-----------------------------------------------|-------------------------------------------|-------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
|                            | 64.45 ±<br>11.98                                     | 65.54 ±<br>12.04                              | 69.56 ±<br>11.71                          | 0.026                   | 0.895                                           | 0.024                                           | 0.224                                           |
| Sex, male                  | 61<br>(81.33%)                                       | 30<br>(85.71%)                                | 64<br>(85.33%)                            | 0.756                   | 0.605                                           | 0.589                                           | 0.88                                            |
| Hypertension               | 50<br>(66.67%)                                       | 19<br>(54.29%)                                | 45 (60%)                                  | 0.435                   | 0.211                                           | 0.411                                           | 0.552                                           |
| Diabetes                   | 30 (40%)                                             | 7 (20%)                                       | 18<br>(23.1%)                             | 0.029                   | 0.039                                           | 0.024                                           | 0.716                                           |
| HbA1c, median<br>(IQR)     | 6.57<br>(6.14-<br>7.0)                               | 5.85(5.56-<br>6.13)                           | 5.68<br>(5.43-<br>5.92)                   | 0.032                   | 0.523                                           | 0.01                                            | 0.159                                           |
| Hyperlipidemia             | 15 (20%)                                             | 6 (17.14%)                                    | 8<br>(10.67%)                             | 0.281                   | 0.722                                           | 0.17                                            | 0.551                                           |
| TG, mmol/L                 | 1.344 ±<br>0.697                                     | 1.333 ±<br>0.651                              | 1.465 ±<br>0.79                           | 0.516                   | 0.997                                           | 0.562                                           | 0.648                                           |
| TC, mmol/L                 | 4.09 ±<br>1.202                                      | 4.073 ±<br>1.391                              | 4.122 ±<br>1.199                          | 0.977                   | 0.998                                           | 0.986                                           | 0.98                                            |
| LDL, mmol/L                | 2.292 ±<br>0.822                                     | 2.498 ±<br>0.949                              | 2.316 ±<br>0.725                          | 0.437                   | 0.428                                           | 0.981                                           | 0.515                                           |
| HDL, mmol/L                | 1.072 ±<br>0.264                                     | 1.001 ±<br>0.298                              | 1.079 ±<br>0.251                          | 0.323                   | 0.393                                           | 0.985                                           | 0.321                                           |
| Coronary<br>artery disease | 18 (24%)                                             | 11<br>(31.43%)                                | 14<br>(18.67%)                            | 0.33                    | 0.41                                            | 0.473                                           | 0.154                                           |
| Smoking                    | 29<br>(38.67%)                                       | 11<br>(31.43%)                                | 22<br>(29.33%)                            | 0.461                   | 0.462                                           | 0.231                                           | 0.835                                           |
| Drinking                   | 12 (16%)                                             | 2 (5.71%)                                     | 7<br>(9.33%)                              | 0.221                   | 0.218                                           | 0.188                                           | 0.718                                           |
| Aspirin                    | 51 (68%)                                             | 29<br>(82.86%)                                | 53<br>(70.67%)                            | 0.259                   | 0.103                                           | 0.87                                            | 0.129                                           |
| Statin                     | 66 (88%)                                             | 32 (91.4%)                                    | 67 (87%)                                  | 0.795                   | 0.591                                           | 0.854                                           | 0.499                                           |

Data are mean ± standard deviation or n (%), unless otherwise stated.

TG: Triglyceride; TC: total cholesterol; LDL: low-density lipoprotein, HDL: high-density lipoprotein; IQR, interquartile range.

| Age, yr                                                                                                                         | Group 1<br>(acute recurrent stroke, n = 75) | Group 2<br>(acute first stroke, n = 35) | Group 3<br>(chronic stroke, n = 78) | $P_{all}$ | $P_{group}$<br>1 vs.<br>group 2 | $P_{group}$<br>1 vs.<br>group 3 | $P_{group}$<br>2 vs.<br>group 3 |
|---------------------------------------------------------------------------------------------------------------------------------|---------------------------------------------|-----------------------------------------|-------------------------------------|-----------|---------------------------------|---------------------------------|---------------------------------|
|                                                                                                                                 | 64.45 ± 11.98                               | 65.54 ± 12.04                           | 69.56 ± 11.71                       | 0.026     | 0.895                           | 0.024                           | 0.224                           |
| Uric acid                                                                                                                       | 307.72 ± 107.15                             | 309.29 ± 81.67                          | 310.32 ± 72.65                      | 0.984     | 0.996                           | 0.982                           | 0.998                           |
| Homocysteine                                                                                                                    | 21.395 ± 14.067                             | 23.793 ± 16.89                          | 24.391 ± 13.787                     | 0.424     | 0.699                           | 0.413                           | 0.978                           |
| Interval time examination, month                                                                                                | 14.7 ± 6.6                                  | -                                       | 16.1 ± 4.2                          | 0.118     | -                               | -                               | -                               |
| Data are mean ± standard deviation or n (%), unless otherwise stated.                                                           |                                             |                                         |                                     |           |                                 |                                 |                                 |
| TG: Triglyceride; TC: total cholesterol; LDL: low-density lipoprotein, HDL: high-density lipoprotein; IQR, interquartile range. |                                             |                                         |                                     |           |                                 |                                 |                                 |

## VWI Characteristics

Left-sided lesions were significantly more common in group 1 than in group 2 (62.67% vs. 37.14%,  $P = 0.012$ ) and group 3 (62.67% vs. 38.67%,  $P = 0.003$ ). Distal occluded vessel lumen hyperintensity was significantly more common in group 1 than in group 2 ( $P = 0.01$ ) and group 3 ( $P < 0.001$ ). Ipsilateral posterior communicating artery (PCoM)  $\geq 4$  was more common in group 3 than in group 1 ( $P = 0.027$ ) and group 2 ( $P < 0.001$ )(Table 2); it was significantly more common in group 1 than in group 2 ( $P = 0.014$ ) (Fig. 2 Upper and Bottom). Ipsilateral meningeal branch  $\geq 2$  scores was more common in group 1 than in group 3. Intraplaque hemorrhage for proximal occlusion was significantly more common in group 2 than in group 1 ( $P = 0.025$ ).(Fig. 2 Middle)

Table 2  
Comparison of image characteristics in the three CICA0 groups

| <b>Left-sided lesion</b>                                                                                                                            | <b>Group 1<br/>(acute recurrent stroke, n = 75)</b> | <b>Group 2<br/>(acute first stroke, n = 35)</b> | <b>Group 3<br/>(chronic stroke, n = 78)</b> | <b><i>P</i><sub>all</sub></b> | <b><i>P</i><sub>group 1 vs. group 2</sub></b> | <b><i>P</i><sub>group 1 vs. group 3</sub></b> | <b><i>P</i><sub>group 2 vs. group 3</sub></b> |
|-----------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------|-------------------------------------------------|---------------------------------------------|-------------------------------|-----------------------------------------------|-----------------------------------------------|-----------------------------------------------|
|                                                                                                                                                     | <b>47<br/>(62.67%)</b>                              | <b>13<br/>(37.14%)</b>                          | <b>29<br/>(38.67%)</b>                      | <b>0.005</b>                  | <b>0.012</b>                                  | <b>0.003</b>                                  | <b>0.894</b>                                  |
| <b>Proximal occlusion</b>                                                                                                                           |                                                     |                                                 |                                             |                               |                                               |                                               |                                               |
| Tapered stump                                                                                                                                       | 43 (57.3%)                                          | 26 (74.3%)                                      | 57 (73.1%)                                  | 0.07                          | 0.087                                         | 0.041                                         | 0.893                                         |
| Intra-plaque hemorrhage                                                                                                                             | 10 (13.3%)                                          | 11 (31.4%)                                      | 14 (17.9%)                                  | 0.074                         | 0.025                                         | 0.433                                         | 0.11                                          |
| Plaque calcification                                                                                                                                | 39 (52%)                                            | 20 (57.1%)                                      | 42 (53.8%)                                  | 0.88                          | 0.614                                         | 0.819                                         | 0.745                                         |
| Lipid necrosis core                                                                                                                                 | 26 (34.67%)                                         | 13 (37.14%)                                     | 25 (33.33%)                                 | 0.926                         | 1                                             | 0.746                                         | 0.795                                         |
| Distal occluded vessel lumen hyperintensity                                                                                                         | 39 (52%)                                            | 9 (25.7%)                                       | 13 (16.7%)                                  | < 0.001                       | 0.01                                          | < 0.001                                       | 0.261                                         |
| FVH                                                                                                                                                 | 46 (61.33%)                                         | 21 (60%)                                        | 18 (24%)                                    | < 0.001                       | 0.894                                         | < 0.001                                       | < 0.001                                       |
| <b>Collateral circulation</b>                                                                                                                       |                                                     |                                                 |                                             |                               |                                               |                                               |                                               |
| Acom score ≥ 4                                                                                                                                      | 40 (53.3%)                                          | 24 (68.6%)                                      | 56 (71.8%)                                  | 0.048                         | 0.131                                         | 0.016                                         | 0.728                                         |
| Ipsilateral Pcom score ≥ 4                                                                                                                          | 28 (37.3%)                                          | 5 (14.3%)                                       | 43 (55.1%)                                  | < 0.001                       | 0.014                                         | 0.027                                         | < 0.001                                       |
| Contralateral Pcom score ≥ 4                                                                                                                        | 22 (29.3%)                                          | 11 (31.4%)                                      | 32 (41%)                                    | 0.289                         | 0.823                                         | 0.13                                          | 0.331                                         |
| Ipsilateral OA score ≥ 4                                                                                                                            | 6 (6%)                                              | 3 (8.6%)                                        | 7 (9%)                                      | 1.00                          | 1.00                                          | 0.83                                          | 1.00                                          |
| Ipsilateral meningeal branch ≥ 2                                                                                                                    | 9 (12%)                                             | 5 (14.3%)                                       | 23 (29.5%)                                  | 0.017                         | 0.764                                         | 0.008                                         | 0.084                                         |
| All data are n (%). Acom: Anterior communicating artery; Pcom: posterior cerebral artery; OA: ophthalmic artery; FVH: FLAIR vascular hyperintensity |                                                     |                                                 |                                             |                               |                                               |                                               |                                               |

## Association between VWI Characteristics and Recurrent Stroke

In univariate analysis, diabetes, occluded proximal intraplaque hemorrhage, left-sided lesion, distal occluded vessel lumen hyperintensity, and ipsilateral Pcom < 4 were significantly associated with stroke recurrence (all  $P < 0.05$ ). In multivariate analysis, the variables independently associated with stroke recurrence were left-sided lesion (OR, 2.54, 95% CI: 1.05–6.14,  $P = 0.04$ ), distal occluded vessel lumen hyperintensity (OR, 2.86; 95% CI: 1.16–7.06;  $P = 0.02$ ), and ipsilateral Pcom < 4 (OR, 3.48, 95% CI: 1.15–10.55,  $P = 0.03$ ) (Table 3). The ROC was used to identify the acute stroke recurrence combined with ipsilateral Pcom scores, distal occluded vessel lumen hyperintensity and left-sided lesion, AUC is 0.838 (95% CI, 0.782–0.894), the sensitivity was 0.853, the specificity was 0.707 (Fig. 3). AUC is 0.714 for ipsilateral Pcom, 0.665 for distal occluded vessel lumen hyperintensity, and 0.616 for left-sided lesion, respectively.

Table 3

Results of univariate and multivariate analysis showing association of different factors with stroke recurrence

|                                              | Univariate Analysis |            |      | Multivariate Analysis |            |      |
|----------------------------------------------|---------------------|------------|------|-----------------------|------------|------|
|                                              | OR                  | 95% CI     | P    | OR                    | 95% CI     | P    |
| <b>Age</b>                                   | 1.17                | 0.52–2.62  | 0.71 | 1.14                  | 0.40–3.21  | 0.81 |
| <b>Male</b>                                  | 1.38                | 0.45–4.18  | 0.57 | 1.37                  | 0.32–5.83  | 0.67 |
| <b>Hypertension</b>                          | 1.68                | 0.74–3.82  | 0.21 | -                     | -          | -    |
| <b>Diabetes</b>                              | 2.67                | 1.03–6.88  | 0.04 | 2.37                  | 0.87–6.51  | 0.09 |
| <b>HbA1c</b>                                 | 2.25                | 0.90–5.61  | 0.08 | 1.44                  | 0.83–5.21  | 0.41 |
| <b>Hyperlipidemia</b>                        | 1.21                | 0.42–3.44  | 0.72 | -                     | -          | -    |
| <b>TC</b>                                    | 2.10                | 0.65–6.83  | 0.22 | -                     | -          | -    |
| <b>LDL</b>                                   | 0.52                | 0.15–1.84  | 0.31 | -                     | -          | -    |
| <b>HDL</b>                                   | 1.15                | 0.45–2.95  | 0.78 | -                     | -          | -    |
| <b>Coronary disease</b>                      | 0.69                | 0.28–1.68  | 0.41 | -                     | -          | -    |
| <b>Smoking</b>                               | 1.38                | 0.59–3.22  | 0.46 | 0.97                  | 0.28–3.38  | 0.96 |
| <b>Drinking</b>                              | 3.14                | 0.66–14.88 | 0.15 | -                     | -          | -    |
| <b>Aspirin use</b>                           | 0.44                | 0.16–1.20  | 0.11 | -                     | -          | -    |
| <b>Statin use</b>                            | 0.69                | 0.17–2.71  | 0.59 | -                     | -          | -    |
| <b>Uric acid</b>                             | 2.24                | 0.59–8.42  | 0.23 | -                     | -          | -    |
| <b>Homocysteine</b>                          | 0.92                | 0.39–2.17  | 0.84 | -                     | -          | -    |
| <b>Left-sided lesion</b>                     | 2.84                | 1.24–6.52  | 0.01 | 2.54                  | 1.05–6.14  | 0.04 |
| <b>Proximal occlusion</b>                    |                     |            |      |                       |            |      |
| Tapered stump                                | 2.15                | 0.89–5.21  | 0.09 | 0.38                  | 0.13–1.10  | 0.08 |
| Intra-plaque hemorrhage                      | 0.34                | 0.13–0.89  | 0.03 | 0.34                  | 0.12–0.99  | 0.05 |
| Plaque calcification                         | 0.81                | 0.36–1.82  | 0.61 | -                     | -          | -    |
| Lipid necrosis core                          | 0.90                | 0.39–2.07  | 0.80 | -                     | -          | -    |
| <b>Distal occlusion lumen hyperintensity</b> | 3.13                | 1.29–7.57  | 0.01 | 2.86                  | 1.16–7.061 | 0.02 |
| <b>FVH</b>                                   | 1.06                | 0.47–2.40  | 0.89 | -                     | -          | -    |

|                               | Univariate Analysis |            |      | Multivariate Analysis |            |      |
|-------------------------------|---------------------|------------|------|-----------------------|------------|------|
|                               | OR                  | 95% CI     | P    | OR                    | 95% CI     | P    |
| <b>Collateral circulation</b> |                     |            |      |                       |            |      |
| Acom                          | 0.52                | 0.22–1.22  | 0.13 | -                     | -          | -    |
| Ipsilateral Pcom              | 3.57                | 1.24–10.28 | 0.02 | 3.48                  | 1.15–10.55 | 0.03 |
| Contralateral Pcom            | 0.91                | 0.38–2.16  | 0.82 | -                     | -          | -    |
| Ipsilateral ophthalmic artery | 0.93                | 0.22–3.95  | 0.92 | -                     | -          | -    |
| Ipsilateral meningeal branch  | 0.82                | 0.25–2.65  | 0.74 | -                     | -          | -    |

## Reproducibility

Inter-reader agreement was good for stump, intraplaque hemorrhage, calcification, lipid-rich necrosis core (for proximal occlusion), distal occluded vessel lumen hyperintensity, Acom score, ipsilateral Pcom score, contralateral Pcom score, ipsilateral ophthalmic artery score, and ipsilateral meningeal branch score; the kappa coefficients were 0.969, 0.837, 0.813, 0.871, 0.942, 0.905, 0.886, 0.891, 0.807, and 0.924, respectively.

## Discussion

This study compared the clinical and VWI characteristics in patients with CICA0 presenting with acute recurrent stroke, acute first stroke, and chronic stroke. Diabetes, intraplaque hemorrhage for proximal occlusion, distal occluded vessel lumen hyperintensity, poor ipsilateral PCom, and left-sided lesion were all significantly associated with CICA0 stroke recurrence. However, in multivariate analysis, the only independent predictors of recurrent stroke were distal occluded vessel lumen hyperintensity, poor ipsilateral PCom, and left-sided lesion.

Symptoms, stroke recurrence risk, and prognosis in CICA0 depend on vascular factors such as occluded vessel distal thrombosis extension, collateral reserve, and cerebral hemodynamic disorders (20). Intraluminal thrombosis in the distal occluded segment has been previously identified as a potential risk factor for recurrent stroke in CICA0 (21). Chronic internal carotid artery occlusion is associated with thrombus formation and extension in the lumen distal to the occlusion as the disease progresses. The histological features of thrombus can reflect the pathological process of CICA0, so as MRI findings of intracranial hemorrhage (22, 23). Corti et al [24] obtained MR images using black-blood T1-weighted and T2-weighted MRI to assess the age of thrombus by assessing different oxygenation states of erythrocytic hemoglobin in the thrombus and changes in intracellular/matrix content of proteins and red cell hydration over time. Hyperintensity of thrombus is considered as recent or fresh thrombus (< 3 weeks), and the stability of thrombus is poor; The lower the thrombus signal is better, it represents the

thrombus organization (> 6 weeks), and the more stable the thrombus[22, 24]. Therefore, we believe that the thrombus is fresh or recent in distal lumen hyperintensity of CICA0 accompanied without enhancement. VWI can also accurately distinguish thrombus between old and fresh thrombus by signal changes. Fresh thrombus forming in the caudal part of the thrombus has a loose structure, and dislodgement of thrombus fragments may cause stroke recurrence. The finding of hyperintensity in the distal occluded vessel lumen in this study supports previous findings that dislodgment of caudal thrombus is one of the most common mechanisms of stroke recurrence in patients with CICA0 (8, 25). Hyperintensity of distal thrombus in the occluded vessel on VWI is an important predictor of ischemic stroke recurrence in patients with CICA0. Therefore, VWI monitoring of signal changes in the distal occluded lumen signal can be helpful for adjusting treatment strategies.

In this study, poor primary collateral circulation (poor ipsilateral PCom) was another VWI imaging feature predictive of ischemic stroke recurrence in CICA0. Previous studies (26–28) have suggested that primary collateral insufficiency could lead to hemodynamic disorder and increase risk of stroke. The present study provides more direct evidence that poor flow in primary collateral vessels—but not secondary collateral vessels—is independently related to CICA0 stroke recurrence. Thus, this study confirmed that the primary collateral circulation plays an active role in compensatory hemodynamics, and that the secondary collateral circulation (reverse blood flow in the ophthalmic artery and increased blood flow within the meningeal collateral) may have limited compensatory capacity and is simply a cerebrovascular response to hemodynamic impairment (29, 30).

Interestingly, this study found that left-sided lesion was an independent predictor of stroke recurrence. Some authors believe that the primary compensatory response to carotid artery obstruction is an increase in contralateral internal carotid artery blood flow, and that the external carotid artery and vertebral artery only have a secondary compensatory role (31). As the brachiocephalic trunk divides into the right common carotid artery and the subclavian artery, occlusion of the left internal carotid artery results in compensatory increased blood flow from the brachiocephalic trunk into the subclavian artery. Moreover, left predominance of the vertebral artery is more common (32). These factors may together cause reduced flow to the brain from the right internal carotid artery system when the left internal carotid artery is occluded. However, this conclusion must be confirmed in a larger study.

Severe stenosis and occlusion of large intracranial vessels will stimulate the formation of cerebral collateral circulation, and therefore the leptomeningeal collateral circulation may show FVH, indicating slow anterograde or retrograde flow (14, 33). Studies on ischemic stroke due to internal carotid artery occlusion have shown microthrombotic signals within meningeal collateral vessels. Reduced perfusion limits the ability of blood flow to flush emboli and microemboli, and so blood flow in the territory where the embolus is blocking the blood supply artery is reduced (29, 34, 35). This is consistent with our results that showed FVH to be significantly more common in patients with acute recurrent stroke and acute first stroke than in patients with chronic stroke. Thus, FVH may be a sign of internal carotid artery occlusion stroke.

This study found that patients with recurrent CICA stroke were more likely to have diabetes and less likely to have occlusive proximal plaque hemorrhage. However, multivariate analysis showed that these factors were not independently related to CICA stroke recurrence; thus, they might be less important reasons for CICA stroke recurrence. A hyperglycemic environment leads to thickening of the capillary basement membrane, aggregation of red blood cells, increase of blood viscosity, and hypercoagulability, all of which increase the risk for recurrent ischemic stroke (36).

There are several limitations in this study. First, this was retrospective single-center observational study with a small sample size, so selection bias is likely. The results of this study need to be confirmed in large longitudinal studies. Second, VWI can evaluate the degree of formation of primary and secondary collateral circulation but it cannot clarify the direction of collateral blood flow.

## Conclusions

To conclude, patients with VWI features of distal occluded vessel lumen hyperintensity, poor ipsilateral primary collateral circulation, and left-sided lesion appear to have increased likelihood of CICA stroke recurrence. VWI can help stratify patients according to risk of recurrence and allow adoption of the appropriate treatment strategy.

## Abbreviations

VWI, Vessel wall imaging; CICA, chronic internal carotid artery occlusion; HbA1c, Hemoglobin A1c; Pcom, posterior communicating; FVH, FLAIR vascular hyperintensity; OR, Odd ratio; CI, Confidence interval; CTA, Computed tomography angiography; MRA, Magnetic resonance angiography; CEMRA, Contrast enhanced magnetic resonance angiography; DSA, Digital subtraction angiography; MRI, Magnetic resonance imaging; T1WI, T1 weighted imaging; T2WI, T2 weighted imaging; FLAIR, Fluid attenuated inversion recovery; DWI, Diffusion weighted imaging; FOV, Field of view; 3D-T1WI-VISTA, Three dimensional T1WI volume isotropic turbo spin-echo acquisition; BB, Black blood; TR, Repetition time; TE, Echo time; NSA, Number of signals averaged; IQR, Interquartile ranges; ROC, Receiver operating characteristic curves; AUC, Area under the curve; TG, Triglyceride; TC, total cholesterol; LDL, Low-density lipoprotein;

HDL, High-density lipoprotein; OA, Ophthalmic artery; FLAIR, Fluid-attenuated inversion recovery; HR-VWI, High resolution vessel wall imaging.

## Declarations

## Ethics approval and consent to participate

This study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics Committee of Shaanxi Provincial People's Hospital ( NO.2023K-S042). All the participants provided

written informed consent. All data used in this study was anonymized and did not involve personal privacy or commercial interests.

## Clinical Trial

Not applicable.

## Competing interests

The authors declare no competing interests.

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## Author Contribution

MT, LL, and NM drafted the manuscript and designed the experiment. JH and XY performed the statistical analysis. MT, LL, and YW collected the data. XZ provided technical support. ZZ contributed to the design of the experiment and also revised the manuscript. All authors contributed to the article and approved the submitted version. LL and MT drafted the manuscript and designed the study. LL performed the statistical analysis. XY and JG contributed to conducting the study and revised the manuscript. MT and KA provided technical support. LL, NM, XS, YN, and YW collected the data. JH, XY and XL helped design the study and revised the manuscript. All authors contributed to the article and approved the submitted version.

## Data availability

The datasets used and analyzed in this study from Shaanxi Provincial People's hospital, Xi'an City, Shaanxi Province, China, and can be obtained from the corresponding author upon reasonable request.

## References

1. Weimar C, Goertler M, Harms L, Diener H-C. Distribution and outcome of symptomatic stenoses and occlusions in patients with acute cerebral ischemia. Arch Neurol (2006) 63(9):1287–91. doi: 10.1001/archneur.63.9.1287.

2. Powers WJ, Derdeyn CP, Fritsch SM, Carpenter DA, Yundt KD, Videen TO, et al. Benign prognosis of never-symptomatic carotid occlusion. *Neurology* (2000) 54(4):878–82. doi: 10.1212/wnl.54.4.878
3. Buslovich S, Hines GL. Spontaneous recanalization of chronic internal carotid artery occlusions: report of 3 cases. *Vasc Endovascular Surg* (2011) 45(1):93–97. doi: 10.1177/1538574410380792.
4. Flaherty ML, Flemming KD, McClelland R, Jorgensen NW, Brown RD Jr. Population-based study of symptomatic internal carotid artery occlusion: incidence and long-term follow-up. *Stroke* (2004) 35(8):e349–52. doi: 10.1161/01.STR.0000135024.54608.3f.
5. Kao H-L, Hung C-S, Li H-Y, Yeh C-F, Huang C-C, Chen Y-H, et al. Long-term outcomes after endovascular recanalization in patients with chronic carotid artery occlusion. *Am J Cardiol* (2018) 122(10):1779–83. doi: 10.1016/j.amjcard.2018.07.049.
6. Lin MS, Chiu MJ, Wu YW, Huang CC, Chao CC, Chen YH, et al. Neurocognitive improvement after carotid artery stenting in patients with chronic internal carotid artery occlusion and cerebral ischemia. *Stroke* (2011) 42(10):2850–4. doi: 10.1161/STROKEAHA.111.613133.
7. Malhotra K, Liebeskind DS. Collaterals in ischemic stroke. *eNeuroscience* (2017) 1(1):1–10. doi: 10.14336/EN.2017.0801.
8. Liberman AL, Zandieh A, Loomis C, Raser-Schramm JM, Wilson CA, Torres J, et al. Symptomatic carotid occlusion is frequently associated with microembolization. *Stroke* (2017) 48(2):394–399. doi: 10.1161/STROKEAHA.116.015375.
9. Rocha M, Delfyett WT, Agarwal V, Aghaebrahim A, Jadhav A, Jovin TG. Diagnostic accuracy of emergency CT angiography for presumed tandem internal carotid artery occlusion before acute endovascular therapy. *J Neurointerv Surg* (2018) 10(7):653–656. doi: 10.1136/neurintsurg-2017-013169.
10. Liu Y, Jia L, Liu B, Meng X, Yang J, Li J, et al. Evaluation of endarterectomy recanalization under ultrasound guidance in symptomatic patients with carotid artery occlusion. *PloS One* (2015) 10(12):e0144381. doi: 10.1371/journal.pone.0144381.
11. Gong Y, Cao C, Guo Y, Chang B, Sheng Z, Shen W, et al. Quantification of intracranial arterial stenotic degree evaluated by high-resolution vessel wall imaging and time-of-flight MR angiography: reproducibility, and diagnostic agreement with DSA. *Eur Radiol* (2021) 31(8):5479–5489. doi: 10.1007/s00330-021-07719-x.
12. Zhang J, Ding S, Zhao H, Sun B, Li X, Zhou Y, et al. Evaluation of chronic carotid artery occlusion by non-contrast 3D-MERGE MR vessel wall imaging: comparison with 3D-TOF-MRA, contrast-enhanced MRA, and DSA. *Eur Radiol* (2020) 30(11):5805–5814. doi: 10.1007/s00330-020-06989-1.
13. Nguyen-Huynh MN, Lev MH, Rordorf G. Spontaneous recanalization of internal carotid artery occlusion. *Stroke* (2003) 34(4):1032–34. doi: 10.1161/01.STR.0000060872.85874.33.
14. Liebeskind DS. Collateral circulation. *Stroke* (2003) 34(9):2279–84. doi: 10.1161/01.STR.0000086465.41263.06.
15. Tang M, Yan X, Gao J, Li L, Zhe X, Zhang X, et al. High-Resolution MRI for Evaluation of the Possibility of Successful Recanalization in Symptomatic Chronic ICA Occlusion: A Retrospective

- Study. *AJNR Am J Neuroradiol* (2022) 43(8):1164–71. doi: 10.3174/ajnr.A7576.
16. Liu LP, Xu AD, Wong LK, Wang DZ, Wang YJ. Expert consensus group of the evaluation & intervention of collateral circulation for ischemic stroke. Chinese consensus statement on the evaluation and intervention of collateral circulation for ischemic stroke. *CNS Neurosci Ther* (2014) 20(3):202–8. doi: 10.1111/cns.12226.
  17. Truong M, Håkansson C, HaileMichael M, Svensson J, Lätt J, Bloch KM, et al. The potential role of T2\*-weighted multi-echo data image combination as an imaging marker for intraplaque hemorrhage in carotid plaque imaging. *BMC Med Imaging* (2021) 21(1):121. doi: 10.1186/s12880-021-00652-x.
  18. Saba L, Yuan C, Hatsukami TS, Balu N, Qiao Y, DeMarco JK, et al. Vessel Wall Imaging Study Group of the American Society of Neuroradiology. Carotid Artery Wall Imaging: Perspective and Guidelines from the ASNR Vessel Wall Imaging Study Group and Expert Consensus Recommendations of the American Society of Neuroradiology. *AJNR Am J Neuroradiol* (2018) 39(2):E9–E31. doi: 10.3174/ajnr.A5488.
  19. Apfaltrer P, Wenz H, Böhme J, Gawlitza M, Groden C, Alonso A, et al. FLAIR Vascular Hyperintensities Indicate Slow Poststenotic Blood Flow in ICA Stenosis. *Clin Neuroradiol* (2021) 31(3):827–831. doi: 10.1007/s00062-020-00941-y.
  20. Thanvi B, Robinson T. Complete occlusion of extracranial internal carotid artery: clinical features, pathophysiology, diagnosis and management. *Postgrad Med J* (2007) 83(976):95–99. doi: 10.1136/pgmj.2006.048041.
  21. Finklestein S, Kleinman GM, Cuneo R, et al. Delayed stroke following carotid occlusion. *Neurology* (1980) 30(1): 84–88. doi: 10.1212/wnl.30.1.84.
  22. Chu B, Kampschulte A, Ferguson MS, Kerwin WS, Yarnykh VL, O'Brien KD, et al. Hemorrhage in the atherosclerotic carotid plaque: a high-resolution MRI study. *Stroke* (2004) 35(5):1079–84. doi: 10.1161/01.STR.0000125856.25309.86.
  23. Furie B, Furie BC. Mechanisms of thrombus formation. *N Engl J Med*, 2008;359(9):938–49. doi: 10.1056/NEJMra0801082.
  24. Corti R, Osende JI, Fayad ZA, et al. In vivo noninvasive detection and age definition of arterial thrombus by MRI. *J Am Coll Cardiol* 2002;39:1366–73. doi: 10.1016/s0735-1097(02)01754-0.
  25. Barnett HJ, Peerless SJ, Kaufmann JC. “Stump” on internal carotid artery – a source for further cerebral embolic ischemia. *Stroke* (1978) 9(5):448–456. doi: 10.1161/01.str.9.5.448.
  26. Reinhard M, Schwarzer G, Briel M, Altamura C, Palazzo P, King A, et al. Cerebrovascular reactivity predicts stroke in high-grade carotid artery disease. *Neurology* (2014) 83(16):1424–1431. doi: 10.1212/WNL.0000000000000888.
  27. Güler S, Utku U, Aynaci O. Early clinical signs, lesion localization, and prognostic factors in unilateral symptomatic internal carotid artery occlusion. *J Stroke Cerebrovasc Dis* (2014) 23(7):1908–1914. doi: 10.1016/j.jstrokecerebrovasdis.2014.02.009.
  28. Silvestrini M, Vernieri F, Pasqualetti P, Matteis M, Passarelli F, Troisi E, et al. Impaired cerebral vasoreactivity and risk of stroke in patients with asymptomatic carotid artery stenosis. *JAMA* (2000)

- 283(16):2122–2127. doi: 10.1001/jama.283.16.2122.
29. Hofmeijer J, Klijn CJ, Kappelle LJ, Van Huffelen AC, Van Gijn J. Collateral circulation via the ophthalmic artery or leptomeningeal vessels is associated with impaired cerebral vasoreactivity in patients with symptomatic carotid artery occlusion. *Cerebrovasc Dis* (2002) 14(1):22–26. doi: 10.1159/000063719.
30. Reinhard M, Müller T, Guschlbauer B, Timmer J, Hetzel A. Dynamic cerebral autoregulation and collateral flow patterns in patients with severe carotid stenosis or occlusion. *Ultrasound Med Biol* (2003) 29(8):1105–1113. doi: 10.1016/s0301-5629(03)00954-2.
31. Kaszczewski P, Elwertowski M, Leszczyński J, et al. Intracranial Flow Volume Estimation in Patients with Internal Carotid Artery Occlusion. *Diagnostics (Basel)* (2022) 12(3):766. doi: 10.3390/diagnostics12030766.
32. Akgun V, Battal B, Bozkurt Y, Oz O, Hamcan S, Sari S, et al. Normal anatomical features and variations of the vertebrobasilar circulation and its branches: an analysis with 64-detector row CT and 3T MR angiographies. *ScientificWorldJournal* (2013) 2013:620162. doi: 10.1155/2013/620162.
33. Legrand L, Tisserand M, Turc G, Naggara O, Edjlali M, Mellerio C, et al. Do FLAIR vascular hyperintensities beyond the DWI lesion represent the ischemic penumbra? *AJNR Am J Neuroradiol* (2015) 36(2): 269–274. doi: 10.3174/ajnr.A4088.
34. Caplan LR, Hennerici M. Impaired clearance of emboli (washout) is an important link between hypoperfusion, embolism, and ischemic stroke. *Arch Neurol* (1998) 55(11):1475–1482. doi: 10.1001/archneur.55.11.1475.
35. Brozici M, van der Zwan A, Hillen B. Anatomy and functionality of leptomeningeal anastomoses: a review. *Stroke* (2003) 34(11):2750–62. doi: 10.1161/01.STR.0000095791.85737.65.
36. Shou, J, Zhou, L, Zhu S, Zhang X. Diabetes is an independent risk factor for stroke recurrence in stroke patients: a meta-analysis. *J Stroke Cerebrovasc Dis* (2015) 24(9):1961–8. doi: 10.1016/j.jstrokecerebrovasdis.2015.04.004.

## Figures

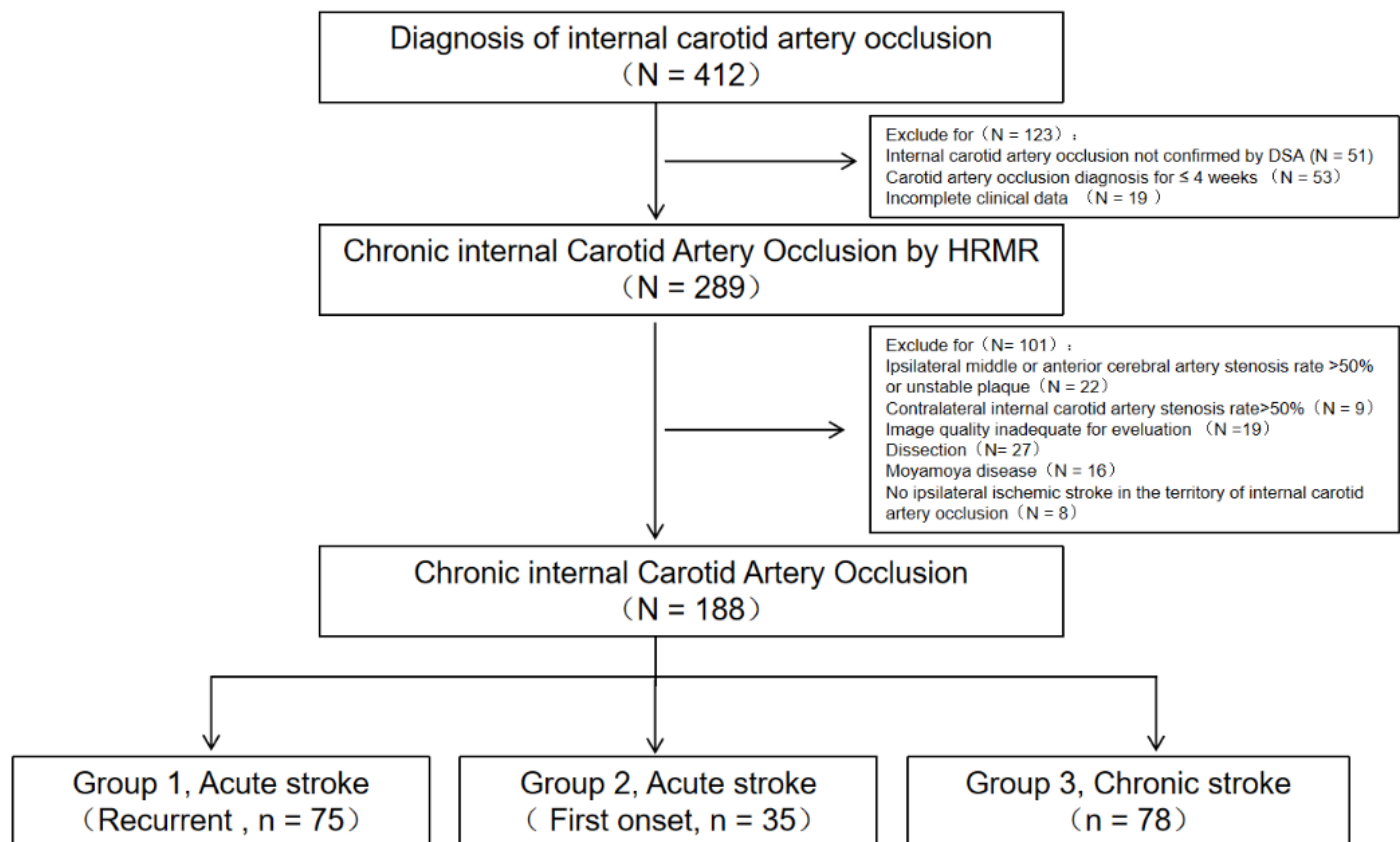


**Figure 1**

Flow chart showing the patient screening process



**Figure 2**

HR-VWI Imaging characteristics in acute recurrent stroke group, acute first stroke group, and chronic stroke group of CICA0. **Upper**, acute recurrent stroke; CEMRA shows left internal carotid artery occlusion (arrow) DWI shows acute recurrent stroke beside the lateral ventricle (arrow) CPR is curved surface reconstruction image on T1WI Vista, and thrombus shows iso-hyperintense in the lumen of occluded segment (arrow); Plaque composition is shown in the proximal occlusion on T1WI and T2WI and CE-T1WI (blue, calcification; orange, hemorrhage; yellow, lipid-rich/necrotic core; purple, loose matrix) The left posterior communicating artery is 3 scores, right is 1 scores on T1WI-Vista (arrow). **Middle**, acute first stroke; CEMRA shows right internal carotid artery occlusion (arrow) DWI shows acute first stroke in frontal lobe (arrow) CPR shows hyperintensity in the proximal occlusion and low signal in the distal occlusion (arrow); Plaque composition is shown in the proximal occlusion on T1WI and T2WI and CE-

T1WI (blue, calcification; orange, hemorrhage; yellow, lipid-rich/necrotic core; purple, loose matrix) The right posterior communicating artery is 1 scores, left is 2 scores on T1WI-Vista (arrow) . **Bottom**, chronic stroke, CEMRA shows right internal carotid artery occlusion (arrow) DWI shows chronic stroke beside the lateral ventricle (arrow) CPR shows hypointensity in the lumen of occluded segment (arrow); Plaque composition is shown in the proximal occlusion on T1WI and T2WI and CE-T1WI (blue, calcification; orange, hemorrhage; yellow, lipid-rich/necrotic core; purple, loose matrix) The right posterior communicating artery is 4 scores, left is 2 scores on T1WI-Vista (arrow).

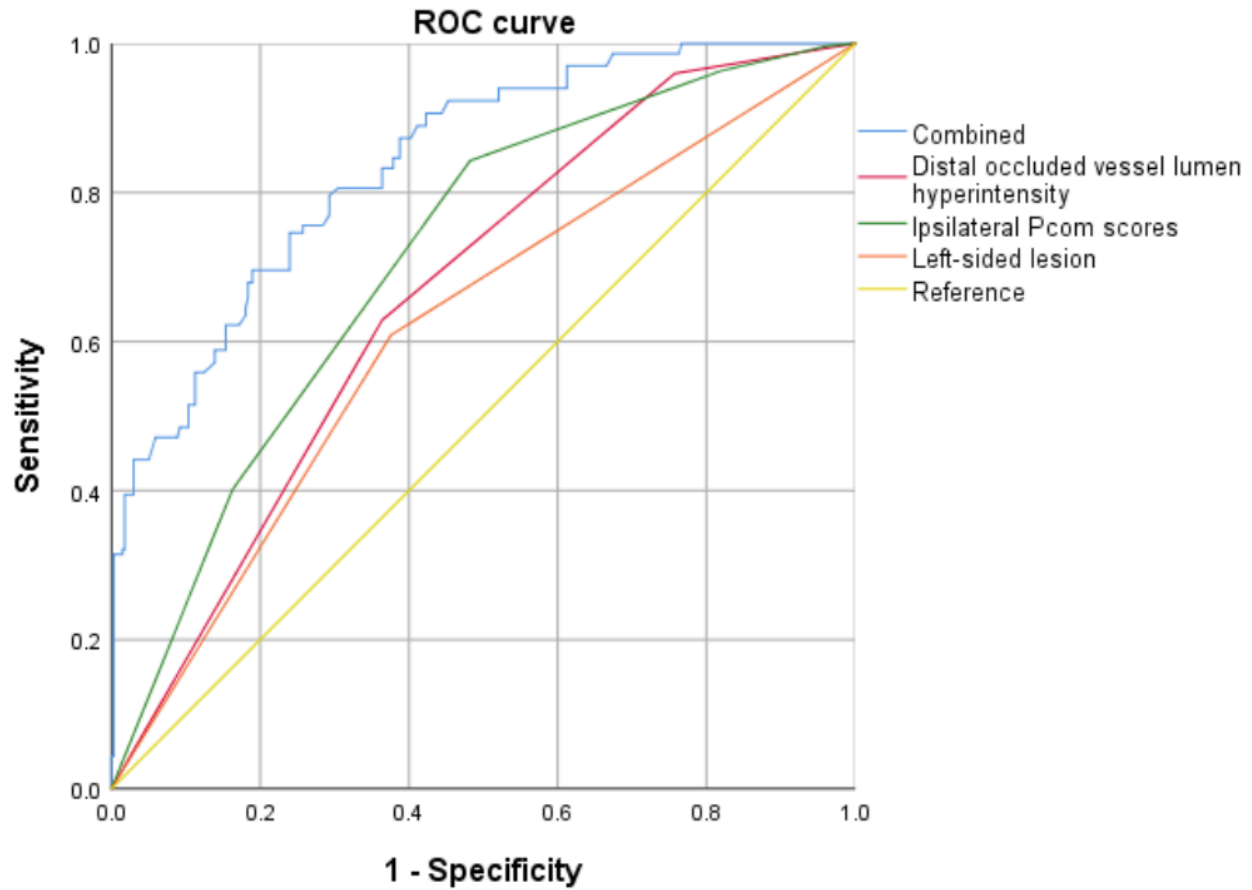


**Figure 3**

ROC for predicting CICA stroke recurrence. The ROC curve analysis showed that the sensitivity (0.853), specificity (0.707), and area under curve (0.838, 95% CI, 0.782-894) of combined detection of ipsilateral Pcom, distal occluded vessel lumen hyperintensity, and left-sided lesion (blue). AUC is 0.714 for ipsilateral Pcom (green), 0.665 for distal occluded vessel lumen hyperintensity red, and 0.616 for left-sided lesion pink.