Volume 24, No 2 | International Journal of Radiation Research, April 2026

Cerebral artery stenosis causing ischemic stroke assessed with high-resolution magnetic resonance vascular-wall imaging (HR-VW-MRI)

F. Zhang[1*], Y. Zhang[2], J. Liu[3], Y. Hu[4], G. Liu[1]

[1]*Department of Radiology, Beijing Geriatric Hospital, Beijing 100095, China*
[2]*Department of Neurology, Beijing Geriatric Hospital, Beijing 100095, China*
[3]*Department of Radiology, Beijing Friendship Hospital, Capital Medical University, Beijing 100050, China*
[4]*Department of Information Center, Beijing Ditan Hospital, Capital Medical University, Beijing 100015, China*

► ***Original article***

Corresponding author:
Fengling Zhang, M.M.,
E-mail:
fengling4097@hotmail.com

Received: June 2025
Final revised: October 2025
Accepted: November 2025

Int. J. Radiat. Res., April 2026; 24(2): 417-422

DOI: 10.61186/ijrr.24.2.17

Keywords: *High-resolution vessel wall magnetic resonance imaging, intracranial atherosclerotic stenosis, ischemic stroke, plaque.*

ABSTRACT

Background: To investigate the impact of plaque characteristics of mild to moderate stenotic middle cerebral artery atherosclerosis (ICAD) on the formation of plaques responsible for ischemic stroke (IS). ***Materials and Methods:*** A retrospective study on patients with mild to moderate stenotic middle cerebral artery atherosclerosis from December 2019 to December 2024. Patients were divided into two cohorts according to whether the plaque was responsible for IS based on high-resolution magnetic resonance vascular-wall imaging (HR-VW-MRI). Propensity score matching was also used to assign patients to plaque and non-plaque IS groups. Univariate and multivariate logistic regression analyses, along with receiver operating characteristic (ROC) curve analysis, were performed to assess the impact of plaque characteristics on IS-related plaque formation. ***Results:*** IS-related plaques had significantly higher volume and steepness than non-IS-related plaques ($P<0.05$), and there were notable differences in remodeling mode and bleeding. Logistic regression showed that the remodeling method (odds ratio (OR) = 0.547), plaque volume (OR=1.092), and plaque steepness (OR=1.089) were independent predictors of IS-related plaque formation. ROC curve analysis showed an area under the curve (AUC) of 0.620 for the remodeling method, 0.718 for plaque volume, and 0.711 for plaque steepness. The combined predictive model achieved an AUC of 0.795. ***Conclusion:*** Plaque remodeling, volume, and steepness are key factors that influence the formation of IS-related plaques. Their joint application could enhance the prediction of such plaques and aid in early detection and treatment.

INTRODUCTION

Intracranial atherosclerosis (ICAD) is the leading cause of ischemic stroke (IS) [1]. Stroke occurs in about 30 to 50% of patients with ICAD in Asian populations [2]. In diagnosing IS, the degree of stenosis mainly indicates etiology and stroke risk. However, luminal stenosis is not a good explanation of the mechanism causing stroke because it may only imply insufficient distal vascular perfusion, as in the case of arterio-arterial embolisms. Thus, the risk needs to be assessed in conjunction with the vessel wall and plaque characteristics [3, 4].

Current routine cerebral arterial screening methods include magnetic resonance angiography (MRA), computed tomography angiography (CTA), and digital subtraction angiography (DSA). DSA offers high sensitivity and specificity and is widely acknowledged as the gold standard for evaluating cerebral arteries [5].

In recent years, high-resolution vessel-wall magnetic resonance imaging (HR-VW-MRI) technology has developed rapidly. It has been widely used in the extracranial part of the carotid and cerebral arteries. It can clearly show the nature of the plaque, the structure of the arterial walls, the characteristics of entrapment, and the characteristics of perivascular lesions [6, 7]. It also shows good agreement with DSA in the measurement of stenosis, which is clinically significant for the diagnosis of ICAD with mild to moderate stenosis and assessment of plaque stability [8].

A previous study compared the characteristics of IS-related and non-IS-related plaques in cases of IS and found some differences in enhancement rate, volume, and distribution characteristics [9, 10]. However, the study did not analyze the correlations with their formation. Our study showcases innovative research by examining the link between plaque characteristics and the development of responsible plaques using HR-VW-MRI. The goal is to offer useful data for predicting the formation of responsible plaques in clinical settings.

[DOI: 10.61186/ijrr.24.2.17]

MATERIALS AND METHODS

Participants and groups

This study retrospectively analyzed 98 patients with mild to moderate stenosis of the middle cerebral artery (MCA) who were admitted to the radiology department from December 2019 to December 2024. The patients were divided into the symptomatic group (n=62) and the asymptomatic group (n=36) according to the presence or absence of symptoms. Table 1 indicates no significant difference in the overall data between the two groups (P>0.05). The inclusion criteria were (1) receiving HR-VW-MRI within 1 week of the onset of symptoms and diagnosed with mild to moderate stenosis of MCA (stenosis rate <69%), (2) over 60 years of age and having a recent cerebral ischemic attack, and (3) complete data about the case and follow-up. The exclusion criteria were (1) a combination of intracranial arterial occlusion and cardiogenic embolism, (2) clinical symptoms not caused by cerebral arterial stenosis, and (3) cases that could not be evaluated due to poor image quality.

Table 1. General Information Comparison [n (%)].

	Symptomatic group(n=62)	Asymptomatic group(n=36)	t/x^2	P
Age (Years)	69.92±4.31	70.19±4.86	-0.290	0.772
Sex (Male/Female)	36(58.06)/26 (41.94)	19(52.78)/17 (47.22)	0.259	0.611
History of Hypertension (Yes/No)	39(62.90)/23 (37.10)	27(75.00)/19 (25.00)	0.197	0.657
History of diabetes (Yes/No)	16(25.81)/46 (74.19)	11(30.56)/25 (69.44)	0.257	0.612
Smoking (Yes/No)	30(48.39)/32 (51.61)	15(41.67)/21 (58.33)	0.414	0.520
Triglyceride (mmol/L)	1.49±0.64	1.46±0.50	0.208	0.836
HDL (mmol/L)	1.23±0.24	1.17±0.24	1.278	0.204
LDL (mmol/L)	2.70±0.75	2.62±0.66	0.497	0.620
Homocysteine (mmol/L)	13.36±3.09	12.72±3.18	0.965	0.337

Patients were divided into two cohorts based on their plaque delineation: an observation screening cohort (IS-related plaque) and a control screening cohort (non-IS-related plaque). The plaques in the two cohorts were later subjected to propensity matching scoring based on various conditions, including stenosis rate, plaque volume, and plaque steepness. The IS-related plaque group and the non-IS-related plaque group were formed after the matching was completed. This study was approved by the Medical Ethics Committee of Beijing Geriatric Hospital (Ethics Approval Number: BJLNYY-2021-018).

HR-VW-MRI examination

The examination was performed using a magnetic resonance imaging scanner (Model: Ingenia 3.0T, Philips (China) Investment Co., Ltd., National Medical Products Administration Imported Medical Device Registration Certificate No. 2015-3282757). Conventional sequences included axial T1-weighted imaging (T1WI), T2-weighted imaging (T2WI), T2 fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), and apparent diffusion coefficient (ADC) mapping. Within 2 weeks of symptom onset, HR-VW-MRI was also performed. The protocol consisted of three-dimensional time-of-flight magnetic resonance angiography (3D-TOF-MRA) followed by pre-contrast black-blood T1WI acquired perpendicular to the M1 segment of the middle cerebral artery. The imaging parameters were as follows: (1) Conventional sequences: T1WI: repetition time (TR) 261 ms, echo time (TE) 2.3 ms; T2WI: TR 3000 ms, TE 80 ms; FLAIR: TR 7000 ms, TE 120 ms; DWI: TR 3338 ms, TE 98 ms. (2) 3D-TOF-MRA: TR 23 ms, TE 3.5 ms, field of view (FOV) 200×200×84 (mm), reconstructed voxel size 0.45×0.68×1.2 (mm), matrix 444×294×140. (3) High-resolution MRI volumetric isotropic turbo spin-echo acquisition (VISTA) T1WI: TR 400 ms, TE 12 ms, FOV 240×240×189 (mm), matrix 268×268×210, isotropic reconstructed voxel size 0.9 mm^3, and a single signal average acquisition time of 4 minutes and 48 seconds.

Image analysis

All images were assessed by two associate physicians with senior-level experience and above. The images were analyzed by a double-blind method, and disagreement was resolved by consultation between the two physicians. The gold standard for image diagnosis is DSA. The image quality was divided into 4 grades according to the signal-to-noise ratio, the display of the structure, and the contour of the lumen and wall. In grade 1, the image could not display the wall structure, and in grade 2, the vessel wall was vaguely visible, but the structure and contour were blurred. In grade 3, most of the wall structure was clear, and only a slight blurring of the local area was observed; in grade 4, the structure of the wall and the contour of the outer wall were clear. Cases with image quality below grade 2 were excluded.

The vessel and lumen boundaries were manually traced at the point of maximal lumen narrowing (MLN) or at the reference lumen level in the image. This allowed for the calculation of the vessel area (VA) and lumen area (LA) of the plaque or reference lumen, as well as the extraction of quantitative metrics such as the stenosis rate, normalized wall index, and remodeling index. The parameters were calculated as follows:

(1) Calculation and grading of stenosis with reference to Samuels' criteria [11] : Stenosis rate=(1-narrowest diameter of stenosis site Ds/proximal widest diameter Dn)×100% (figure 1A-C).
(2) Normal wall index (NWI)=(wall area at the

[DOI: 10.61186/ijrr.24.2.17]

narrowest point/lumen area at the narrowest point) ×100%.
(3) Remodeling index (RI)=vessel area at the narrowest level/vessel area at the reference level. PR: RI ≥1.05; NR: RI < 0.95 [12].

IS-related plaque determination was performed in cases that had a mild to moderate rate of stenosis. If only one plaque was on the same side as the cerebral infarction, it was considered IS-related plaque. If there was more than one plaque on the ipsilateral side of the cerebral infarction, the plaque with the narrowest lumen was also determined to be IS-related plaque. Patch characteristics and their quantitative and qualitative assessments were calculated as follows:

(1) Patch volume =$\sum_N$(Tube wall area-Lumen area) ×Number of layers occupied by plaques.
(2) Intraplaque hemorrhage (IPH): high signal in HR-VW-MRI and signal intensity 1.5 times higher than that of adjacent tissue.
(3) Plaque distribution: eccentric plaque orientation delineation is performed through the superior, inferior, ventral, or dorsal aspect of the vessel, and if the plaque is located in more than 2 quadrants, the distribution location needs to be recorded in its entirety.
(4) Plaque steepness = plaque height/longitudinal length.
(5) Plaque surface irregularity refers to the discontinuity of the surface of the plaque close to the vessel lumen.

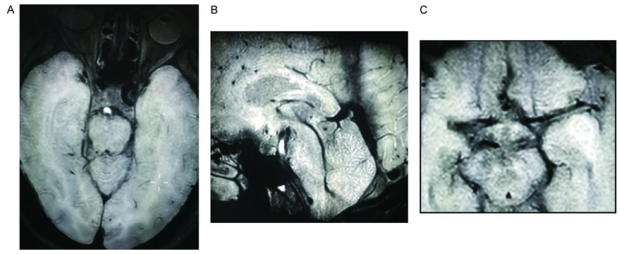

Figure 1. A: HR-VW-MRI sagittal view; **B:** HR-VW-MRI transverse view; **C:** HR-VW-MRI sagittal view. Note: A, B: Severe stenosis of the basilar artery lumen. C: Moderate stenosis of the left middle cerebral artery lumen.

Statistical analysis

The software SPSS 27.0 was used to analyze the data. Continuous variables with a normal distribution were shown as x ± SD, and an independent-sample t-test compared groups. Count data were presented as n (%), and intergroup comparisons were performed using x^2 tests. The bias in plaque characteristics between the symptomatic and asymptomatic populations was eliminated by matching the plaques in the two cohorts based on propensity scores. After matching, the plaques of the two groups were analyzed and compared with those of the two cohorts. Single-factor and multifactorial logistic regression analyses were conducted to examine the factors affecting the presence of IS-related plaques. ROC curves assessed each factor's link to the model's diagnostic effectiveness. P<0.05 was considered statistically significant.

RESULTS

Plaque inclusion

To eliminate the bias in plaque characteristics between symptomatic and asymptomatic patients, plaques from patients in the two cohorts were matched based on propensity scores. A total of 129 plaques were included after successful matching, including 62 IS-related plaques and 67 non-IS-related plaques.

Comparison of plaque characteristics

The plaque characteristics of both groups were examined and compared (table 2). The results revealed significant differences in the remodeling mode, plaque volume, plaque steepness, and plaque hemorrhage (P<0.05). The plaque volume and steepness of IS-related plaques were significantly higher than those of non-IS-related plaques (P<0.05).

Table 2. Comparison of culprit and non-culprit patch characteristics [n (%)].

	Culprit plaque(n=62)	Non-culprit plaque(n=67)	t/x^2	P
Outer wall area(mm^2)	9.17±3.35	8.43±2.77	1.364	0.175
Rate of stenosis (%)	42.68±9.55	40.83±10.53	1.044	0.299
Normal wall index	0.82±0.03	0.81±0.03	0.916	0.361
Reconstruction index	1.00±0.18	0.95±0.15	1.410	0.161
Reconstruction method			6.700	0.035
Positive reconstruction	36(58.06)	25(37.31)		
Negative reconstruction	20(32.26)	27(40.30)		
Neutral reconstruction	6(9.68)	15(22.39)		
Distribution of plaques			0.697	0.952
Upper wall	13(20.97)	16(23.88)		
Lower wall	4(6.45)	6(8.96)		
Front wall	22(35.48)	24(35.82)		
Back wall	10(16.13)	9(13.43)		
Circumferential	13(20.97)	12(17.91)		
Plaque volume(mm^2)	36.75±7.69	30.46±8.19	4.484	0.001
Plaque steepness (%)	31.00±8.96	24.61±7.28	4.461	0.001
Plaque hemorrhage (Yes/No)	29(46.77)/33 (53.23)	17(25.37)/50 (74.63)	6.428	0.011
Plaque surface Irregularities (Yes/No)	30(48.39)/32 (51.61)	26(38.81)/41 (61.19)	1.203	0.273

Single-factor and multi-factor logistic regression analyses

Since the reconstruction method, volume, steepness, and bleeding were significantly different in the previous analysis (P<0.05), they were part of the one-way regression analysis. The results showed that each of the four indicators was significantly different (P<0.05). A multifactorial regression analysis was performed on these four indicators, which showed

[DOI: 10.61186/ijrr.24.2.17]

that reconstruction method (OR=0.547), plaque volume (OR=1.092), and plaque steepness (OR=1.089) were independent factors for IS-related plaque formation (table 3).

Table 3. Factors influencing liability plaque formation via univariate and multivariate logistic regression.

	Single-factor logistic regression			Multi-factor logistic regression		
	OR	95% CI	*P*	OR	95% CI	*P*
Reconstruction method	0.524	0.318~ 0.863	0.011	0.547	0.308~ 0.791	0.039
Plaque volume	1.105	1.051~ 1.161	0.001	1.092	1.036- 1.152	0.001
Plaque steepness	1.102	1.050~ 1.156	0.001	1.089	1.033- 1.147	0.001
Plaque hemorrhage	2.585	1.230~ 5.432	0.012	2.167	0.918- 5.115	0.078

ROC curves

The significant factors in the multifactor regression were used as predictors to establish a prediction model: Z = -4.629-0.561 × reconstruction method + 0.090 × plaque volume + 0.089 × plaque steepness. The three influencing factors and the prediction model were analyzed using ROC curves (figure 2). The results showed that the areas under the curves (AUCs) for the reconstruction method, plaque volume, and plaque steepness were 0.620 (95%CI: 0.524-0.717), 0.718 (95%CI: 0.630-0.807), and 0.711 (95%CI: 0.622-0.799), respectively. The AUC for the prediction model was 0.795 (95% CI :0.718-0.871).

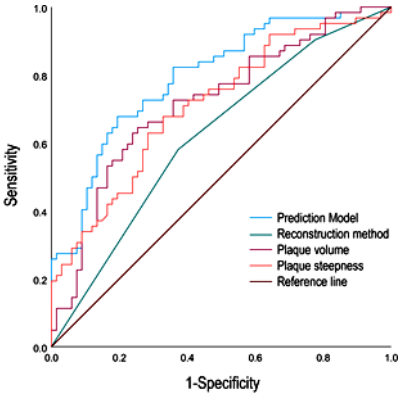

Figure 2. ROC curve. Note: 1-Specificity: false positive rate; sensitivity: true positive rate.

DISCUSSION

IS is the most common type of cardiovascular and cerebrovascular disease, and its incidence increases every year [13]. With the rapid development of high-resolution MRI, it has been gradually applied to the diagnosis of cerebrovascular diseases [14]. HR-VW-MRI utilizes black-blood technology to eliminate blood-flow artifacts, suppress blood-flow signals, and clearly visualize the state of lumen lesions and their pathological development process [15]. This imaging technique provides a more accurate diagnosis of cerebral arterial lesions.

HR-VW-MRI scan sequences include T1WI, T2WI, and PDWI. Currently, the T1WI sequence is more commonly used in clinical applications [7]. It can suppress signals from both blood and cerebrospinal fluid simultaneously, resulting in a clear display of the vessel walls. It can also be used for the assessment of contrast enhancement. PDWI has a higher signal-to-noise ratio and can display black blood [16], but its signal suppression of cerebrospinal fluid is insufficient. T2WI shows black blood and a high signal for cerebrospinal fluid and has some clinical applications in imaging patients with contraindications for gadolinium contrast [17].

Increasing evidence suggests that the induction of IS is caused by changes in the properties of the vascular wall plaques [18]. Therefore, this study correlated the plaque characteristics of mild to moderate stenosis and independent factors that affect plaque formation with IS liability based on imaging with HR-VW-MRI. The study found significant differences in remodeling mode and hemorrhage of IS -related plaques (P<0.05), with their steepness and volume notably greater than non-IS-related plaques (P<0.05).

Arterial remodeling is a compensatory phenomenon of the vessel walls during the formation of atherosclerotic plaques. Positive and negative remodeling are respectively caused by the expansion and contraction of the outer elastic plate area at the site of a lesion [19], which usually occurs in the presence of atherosclerotic plaques. Enlarged arteries provide more blood flow, which is accompanied by compensatory expansion of the walls, vascular fragility, and increased plaque compliance. The rate of positive remodeling in IS-related plaques was 58.06%, whereas it was only 37.31% in non-IS-related plaques. The mechanism of IS symptoms caused by vessel wall remodeling remains unclear. Previous studies have investigated the relationship between remodeling modality and acute IS in patients with MCA stenosis using high-resolution MRI. Results indicate that positive remodeling is unsafe and can cause acute IS [20].

The hemorrhage rate of IS-related plaques was 46.77%, which was higher than that of non-IS-related plaques (25.37%). Intra-plaque hemorrhage was caused by rupture of neovascularization, which increased wall shear stress on the plaque surface, contributing to instability [21]. Plaque hemorrhage has been shown to increase the risk of cerebrovascular events [22]. It has also been shown that the remodeling pattern of the vessel wall correlates with hemodynamics and that positive remodeling is associated with higher ratios of wall shear stress [23].

The steepness of IS-related plaques was significantly higher than that of non-IS-related plaques (P<0.05). Plaque steepness corresponds to the longitudinal curvature of the plaque in the vessel wall and represents the wall shear stress. High wall shear stress subjects' plaques to a greater impact from blood flow and is associated with core necrosis, calcification, and fibrosis, which may lead to fragile

[DOI: 10.61186/ijrr.24.2.17]

plaques that are more prone to dislodgement [24, 25].

Plaque volume was one of the first plaque characteristics to be applied in the study of carotid and coronary arteries. Larger plaques increase the wall shear stress, while lipid aggregation and oxidation within them make plaques less stable. It has been demonstrated that plaque volume correlates with symptom onset and recurrence in patients with mild to moderate stenosis in the MCA [26]. The reason for this may be that larger plaques are more likely to block arteries and rupture, leading to arterial embolism.

CONCLUSION

We confirmed that plaque steepness, volume, and remodeling mode are key factors influencing IS-related plaque formation in patients with mild to moderate MCA stenosis. The prediction model we created demonstrated good accuracy and can support clinical identification of IS-related plaque development. The combined use of these three plaque characteristics in clinical analysis could enhance the prediction of liability plaque formation and strengthen data support for early detection and treatment.

Acknowledgment: None.

***Consent to Publish*:** The manuscript has neither been previously published nor is under consideration by any other journal. The authors have all approved the content of the paper.

***Consent to participate*:** We secured a signed informed consent form from every participant.

***Funding*:** None.

***Conflicts of Interest*:** The authors declare that they have no financial conflicts of interest.

***Ethical consideration*:** This study was approved by the Medical Ethics Committee of Beijing Geriatric Hospital (Ethics Approval Number: BJLNYY-2021-018).

***Author Contribution*:** F.Z., developed and planned the study, performed experiments, and interpreted results. Edited and refined the manuscript with a focus on critical intellectual contributions. Y.Z., participated in collecting, assessing, and interpreting the date. Made significant contributions to date interpretation and manuscript preparation. J.L., Y.H., G.L.; Provided substantial intellectual input during the drafting and revision of the manuscript.

REFERENCES

1. Perera KS, Sharma MA, Eikelboom JW, *et al.* (2025) Combination antithrombotic therapy for reduction of recurrent ischemic stroke in intracranial atherosclerotic disease. *Stroke*, ***56**: 380-389.*
2. Song JY and Kwon SU (2025) Intracranial atherosclerotic stenosis. *Cerebrovasc Dis Extra*, ***15**: 62-67.*
3. Lv Y, Ma X, Zhao W, *et al.* (2024) Association of plaque characteristics with long-term stroke recurrence in patients with intracranial atherosclerotic disease: A 3d high-resolution mri-based cohort study. *Eur Radiol*, ***34**: 3022-3031.*
4. Gutierrez J, Turan TN, Hoh BL, *et al.* (2022) Intracranial atherosclerotic stenosis: Risk factors, diagnosis, and treatment. *Lancet Neurol*, ***21**: 355-368.*
5. Santarosa C, Cord B, Koo A, *et al.* (2020) Vessel wall magnetic resonance imaging in intracranial aneurysms: Principles and emerging clinical applications. *Interv Neuroradiol*, ***26**: 135-146.*
6. Li S, Tang M, Zhang D, *et al.* (2023) The prevalence and prognosis of asymptomatic intracranial atherosclerosis in a community-based population: Results based on high-resolution magnetic resonance imaging. *Eur J Neurol*, ***30**: 3761-3771.*
7. Tian X, Shi Z, Wang Z, *et al.* (2023) Characteristics of culprit intracranial plaque without substantial stenosis in ischemic stroke using three-dimensional high-resolution vessel wall magnetic resonance imaging. *Front Neurosci*, ***17**: 1160018.*
8. Guo Y, Canton G, Baylam Geleri D, *et al.* (2024) Plaque evolution and vessel wall remodeling of intracranial arteries: A prospective, longitudinal vessel wall mri study. *J Magn Reson Imaging*, ***60**: 889-899.*
9. Li M, Song X, Wei Q, *et al.* (2024) The relationship between intracranial atherosclerosis and white matter hyperintensity in ischemic stroke patients: A retrospective cross-sectional study using high-resolution magnetic resonance vessel wall imaging. *Quant Imaging Med Surg*, ***14**: 6002-6014.*
10. Zheng W, Yan H, Tian D, *et al.* (2024) Plaque enhancement of middle cerebral artery and pre-stroke diet are associated with prognosis of subacute ischemic stroke: A prospective high-resolution mr vessel wall imaging study. *Eur J Radiol*, ***180**: 111693.*
11. Samuels OB, Joseph GJ, Lynn MJ, *et al.* (2000) A standardized method for measuring intracranial arterial stenosis. *AJNR Am J Neuroradiol*, ***21**: 643-6.*
12. Luo J, Bai X, Tian Q, *et al.* (2023) Patterns and implications of artery remodeling based on high-resolution vessel wall imaging in symptomatic severe basilar artery stenosis. *Quant Imaging Med Surg*, ***13**: 2098-2108.*
13. Yu J, Chen Q, Dou X, *et al.* (2025) Network pharmacology analyses and corresponding validation of the mechanistic effects of yangyin tongnao granules in ischemic stroke. *Sci Rep*, ***15**: 28074.*
14. Wang J, Liu Y, Tian P, *et al.* (2023) Exosomal circscmh1/mir-874 ratio in serum to predict carotid and coronary plaque stability. *Front Cardiovasc Med*, ***10**: 1277427.*
15. Song Y, Zhou J, Tan Y, *et al.* (2024) Risk factors and clinical significance of ultra-long-term microischemia after intracranial aneurysm embolization. *Neurol Ther*, ***13**: 1173-1190.*
16. Yadav KL, Gupta A, Swarnkar CP, *et al.* (2024) Role of vessel wall magnetic resonance imaging in the diagnosis of intracranial vasculopathies. *Cureus*, ***16**: e73714.*
17. Chai C, Cui Y, Li M, *et al.*(2025) Association between serum uric acid levels and white matter hyperintensities in patients with mild ischemic stroke. *Front Neurol*, ***16**: 1590408.*
18. Kim HJ, Choi EH, Chung JW, *et al.* (2020) Luminal and wall changes in intracranial arterial lesions for predicting stroke occurrence. *Stroke*, ***51**: 2495-2504.*
19. Song X, Li S, Du H, *et al.* (2022) Association of plaque morphology with stroke mechanism in patients with symptomatic posterior circulation icad. *Neurology*, ***99**: e2708-e2717.*
20. Zhang DF, Chen YC, Chen H, *et al.* (2017) A high-resolution MRI study of relationship between remodeling patterns and ischemic stroke in patients with atherosclerotic middle cerebral artery stenosis. *Front Aging Neurosci*, ***9**: 140.*
21. Lee UY and Kwak HS (2021) Evaluation of plaque vulnerability via combination of hemodynamic analysis and simultaneous non-contrast angiography and intraplaque hemorrhage (snap) sequence for carotid intraplaque hemorrhage. *J Pers Med*, ***11**: 856.*
22. Che F, Mi D, Wang A, *et al.* (2022) Extracranial carotid plaque hemorrhage predicts ipsilateral stroke recurrence in patients with carotid atherosclerosis - a study based on high-resolution vessel wall imaging mri. *BMC Neurol*, ***22**: 237.*
23. Liu Y, Li S, Liu H, *et al.* (2025) Clinical implications of haemodynamics in symptomatic intracranial atherosclerotic stenosis by computational fluid dynamics modelling: A systematic review. *Stroke Vasc Neurol*, ***10**: 16-24.*
24. Hakim D, Pinilla-Echeverri N, Coskun AU, *et al.* (2023) The role of endothelial shear stress, shear stress gradient, and plaque topography in plaque erosion. *Atherosclerosis*, ***376**: 11-18.*
25. Woo HG, Kim HG, Lee KM, *et al.* (2023) Wall shear stress associated with stroke occurrence and mechanisms in middle cerebral artery atherosclerosis. *J Stroke*, ***25**: 132-140.*

[DOI: 10.61186/ijrr.24.2.17]

26. Wu G, Wang H, Zhao C*, et al.* (2022) Large culprit plaque and more intracranial plaques are associated with recurrent stroke: A case-control study using vessel wall imaging. *AJNR Am J Neuroradiol*, ***43****: 207-215.*

[DOI: 10.61186/ijrr.24.2.17]