SSR Institute of International Journal of Life Sciences
ISSN (O): 2581-8740 | ISSN (P): 2581-8732
Bansod *et al*., 2026
doi: 10.21276/SSR-IIJLS.2026.12.1.35

Original Article

open access

Atherosclerotic Plaque Characteristics on Vessel Wall Magnetic Resonance Imaging and Their Association with Recurrent Ischemic Stroke: A Systematic Review

Amrit Bansod[1]*, Tushar Patil[2], Lalit Nirwan[3], Harshwardhan Jain[3], S Agrawal[4]

[1]Assistant Professor, Department of Neurology, Super Speciality Hospital and Government Medical College and Hospital, Nagpur, Maharashtra, India

[2]Professor, Department of Neurology, JNMC, Datta Meghe Institute of Higher Education and Research, Wardha, Maharashtra, India

[3]Consultant, Department of Radiology, Tesla MRI and Intervention Centre, Nagpur, Maharashtra, India

[4]Medical Officer, ESIC Medical College and Hospital, Basaidarapur, New Delhi, India

***Address for Correspondence:** Dr. Amrit Bansod, Department of Neurology, Super Speciality Hospital and Government Medical College and Hospital, Nagpur, Maharashtra- 440003, India
E-mail: amritnamratabansod@gmail.com

Received: 23 Aug 2025/ Revised: 19 Oct 2025/ Accepted: 14 Dec 2025

ABSTRACT

Background: Recurrent stroke is associated with significant morbidity and disability. With the advent of modern imaging techniques, focus shifted beyond stenosis to plaque characteristics and vulnerability as the major underlying causes. However, the clinical guidelines still use stenosis as the major criterion. As studies on this topic are scarce, the present systematic review was conducted to assess the association between plaque characteristics and recurrent stroke.

Method: Seven retrospective and prospective studies published in the last 10 years and meeting the inclusion and exclusion criteria were included. The studies included a total of 749 patients. The studies were included after a thorough search of various databases (PubMed, Embase, Cochrane Library, and ScienceDirect).

Results: The majority of the studies included in the review showed a significant association between plaque burden and recurrent stroke, with three studies showing reduced survival on Kaplan-Meier analysis. Recurrent stroke was also found to be significantly associated with intraplaque hemorrhage and increased enhancement ratio.

Conclusion: The present systematic review highlights the importance of plaque characteristics, especially plaque vulnerability and inflammation, in the causation of recurrent stroke. It is recommended that further studies be conducted to develop clinical scores that incorporate plaque characteristics to improve the prediction of recurrence.

Key-words: High-risk plaque, Intracranial atherosclerosis, Magnetic resonance vessel wall imaging, Plaque characteristics, Recurrent stroke, Systematic review

INTRODUCTION

Stroke accounted for 11.60% of the mortality burden worldwide in 2019 [1]. Owing to its high associated morbidity, it is the second leading cause of global death and disability [2].

How to cite this article
Bansod A, Patil T, Nirwan L, Jain H, Agrawal S. Atherosclerotic Plaque Characteristics on Vessel Wall Magnetic Resonance Imaging and Their Association with Recurrent Ischemic Stroke: A Systematic Review. SSR Inst Int J Life Sci., 2026; 12(1): 9217-9224.

Access this article online
https://iijls.com/

It is estimated that the annual new case burden of stroke is about 12.20 million, with a 1-year recurrence rate of 11.10% and a 12-year recurrence rate of 39.70% amongst the survivors [2,3]. Recurrence of stroke is associated with significantly more damage, leading to a higher mortality burden along with poor functional prognosis amongst the survivors as compared to the first-time stroke. It also places significant strain on scarce healthcare services [4].

Intracranial atherosclerotic disease is the leading cause of ischemic stroke. In Asian populations, it may account for up to 30-50% of the ischemic strokes [5,6]. It also poses

doi: 10.21276/SSR-IIJLS.2026.12.1.35

a greater risk of recurrence of the stroke despite aggressive medical management. Therefore, disease risk stratification is crucial for preventing recurrent attacks [7].

Traditionally, stenosis was considered the major underlying factor for stroke. The degree of stenosis is a well-recognized predictor of recurrent stroke. Patients with either more than 50% stenosis or those with less than 50% stenosis but also having vulnerable plaques are at particular risk and aggressive medical therapy is recommended for such cases [8,9]. With the advent of high-resolution vessel wall imaging (HR-VWI), in vivo visualization of plaque characteristics, including vulnerable features such as intraplaque hemorrhage, outward remodeling, and neurovasculature, has become possible. This has provided invaluable insight into plaque vulnerability being a more important underlying cause of recurrent stroke rather than the degree of stenosis alone [10].

Using whole-brain HR-VWI, multiple lesions can be evaluated simultaneously, reducing time [11]. Methodological advances have enabled the routine use of 3 Tesla magnetic resonance imaging (MRI) for VWI, due to its superior spatial resolution and signal-to-noise ratio compared to 1.5 Tesla systems. Optimized sequences, such as 3D T1-weighted sequences, have improved vessel-to-lumen contrast, while consensus guidelines recommend standardized acquisition protocols and delayed post-contrast imaging (~9 minutes) to improve reproducibility [12].

However, the precise role of plaque characteristics in the recurrence of stroke remains understudied, and the present treatment guidelines rely solely on the degree of stenosis. Therefore, the present systematic review was conducted to evaluate the association between various plaque characteristics and stroke recurrence.

MATERIALS AND METHOD

Research Design- The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement was followed during the study. An Institutional Ethics Committee was not required because no human participants were included. PROSPERO registration was not required as the review did not include any intervention.

Search Strategy- An electronic search was performed across various databases: PubMed, Embase, Cochrane Library, and ScienceDirect. The Medical Subject Headings (MeSH) keywords were used for intracranial atherosclerosis, plaque, vessel wall MRI, recurrent stroke, disease progression, and temporal evolution. A manual selection process was used to select the studies, as outlined in the flowchart in Fig. 1.

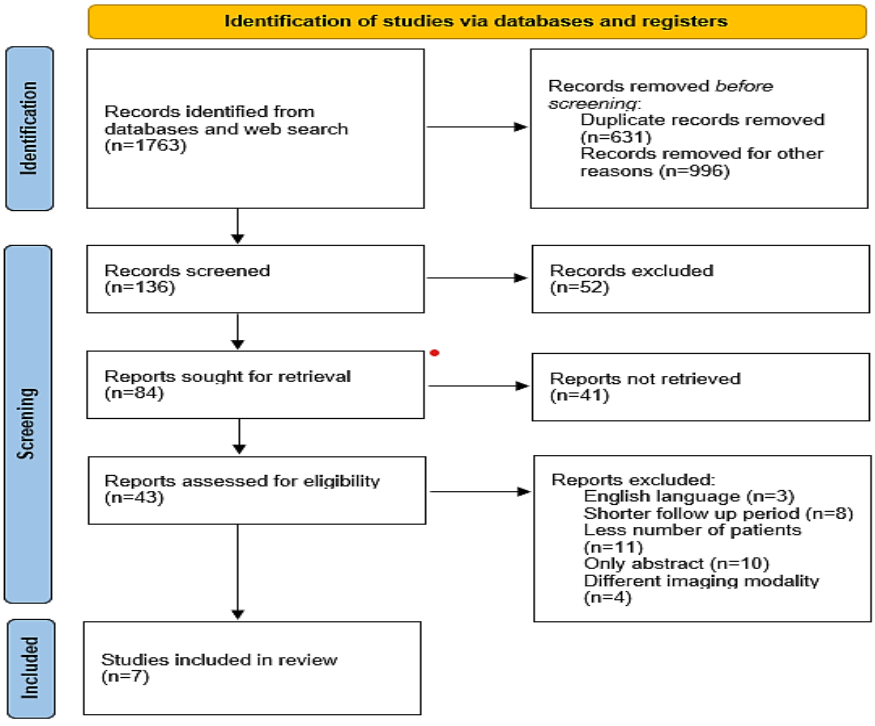

Fig. 1: PRISMA 2020 flow diagram

doi: 10.21276/SSR-IIJLS.2026.12.1.35

Inclusion criteria

- ✓ Studies involving patients aged 18 or older.
- ✓ Prospective or retrospective cohort studies with at least 6 months of follow-up and comparing findings of recurrent ischemic stroke with first-time stroke.
- ✓ Studies with more than 50 patients.
- ✓ Studies where MRI was performed using 3Tesla MRI.
- ✓ Studies published in the last 10 years (after 2016).
- ✓ Studies with full-text publications.
- ✓ English language studies.

Exclusion criteria

- ✓ Studies older than 10 years.
- ✓ Studies with a follow-up period of less than 6 months.
- ✓ Case reports, case series, randomized controlled trials, and reviews.
- ✓ Studies with stroke of other or uncertain etiology.

Statistical Analysis- For the present systematic review, the relevant findings of all the included studies were extracted and summarized in a standard format. A narrative review was conducted, focused on the relevant parameters. For all included studies, $p<0.05$ was considered statistically significant.

RESULTS

In the present systematic review, a total of 1763 studies were retrieved through the various databases (Fig. 1). 136 records were screened, of which 43 were assessed for eligibility. Based on inclusion and exclusion criteria, 36 records were excluded and the remaining 7 studies were included for review (Table 1).

Table 1: Summary of the studies

Study	Year	Type of study	Study population	Findings
Lu *et al.* [13]	2018	Prospective	63	Stroke recurrence was significantly associated with intraplaque hemorrhage, fibrous cap rupture and progression of carotid plaque/change in vessel wall volume [Hazard ratio: 6.05 (1.44-25.51); p=0.01, Hazard ratio: 6.72 (1.37-32.95); p=0.02 and Hazard ratio: 1.19 (1.03-1.37); p=0.02, respectively]. When assessed according to plaque progression, they reported that the Kaplan-Meier curve for stroke recurrence showed higher event-free survival among patients with non-progression of plaque compared with those with plaque progression; p=0.02.
Ran *et al.* [14]	2020	Retrospective	105	Stroke recurrence was significantly associated with plaque burden [Odds ratio: 2.26 (1.03-4.96); p=0.04]. Area under curve was 0.70 for plaque burden.
Shi *et al.* [15]	2021	Prospective	58	Stroke recurrence was significantly associated with progression of plaque burden [Hazard ratio: 6.29 (1.62-24.44); p<0.01]. The Kaplan-Meier curve for recurrence showed that patients without progression of plaque burden had significantly higher event-free survival than those with progression (p<0.05).

doi: 10.21276/SSR-IIJLS.2026.12.1.35

Shen *et al.* [16]	2022	Retrospective	67	Stroke recurrence was significantly associated with change in plaque burden and progression of plaque burden [Odds ratio: 1.11 per 1% increase (1.01-1.22); p=0.03 and Odds ratio: 6.08 (1.51-24.47); p=0.01, respectively].
van Dam-Nolen *et al.* [17]	2022	Prospective	238	Stroke recurrence was significantly associated with intraplaque hemorrhage [Hazard ratio: 2.12 (1.02-4.44); p<0.05 and Hazard ratio: 1.07 (1.00-1.15); p<0.05, respectively]. Kaplan-Meier analysis showed that stroke recurrence was significantly higher in patients with intraplaque hemorrhage as compared to those without hemorrhage in ipsilateral carotid plaque (23% and 11%, respectively; p<0.01).
Lv *et al.* [18]	2024	Prospective	132	Stroke recurrence was significantly associated with plaque burden and enhancement ratio [Hazard ratio: 3.15 (1.34-7.42); p<0.01 and Hazard ratio: 2.17 (1.27-3.70); p<0.01, respectively]. The areas under the curves for predicting recurrence were 0.72 (95% confidence interval: 0.62-0.82) and 0.69 (95% confidence interval: 0.59-0.79) for plaque burden and enhancement ratio, respectively. The Kaplan-Meier curve showed an increased risk of recurrence at cut-off values of 89.20% plaque burden and an enhancement ratio greater than or equal to 0.50; p<0.01.
Yao *et al.* [19]	2025	Prospective	86	Stroke recurrence was significantly associated with progression of plaque burden and progression of enhancement ratio [Hazard ratio: 3.82 (1.12-13.05); p=0.03 and Hazard ratio: 6.14 (1.48-25.51); p=0.01, respectively]. Additionally, they also reported an association between stroke recurrence and vessel expansion [Hazard ratio: 5.17 (1.07-24.83); p=0.04].

doi: 10.21276/SSR-IIJLS.2026.12.1.35

Among the seven studies, most were prospective, and the others were retrospective. A total of 749 patients were included in 7 studies. All studies assessed features of plaque as visualized on 3 Tesla MRI. The plaque characteristics amongst the patients having recurrent stroke were compared with those of the patients having non-recurrent stroke in the cohort. Kaplan-Meier analysis was done in four studies.

It was observed that plaque burden or plaque progression was the most commonly studied parameter (six of seven studies). The studies reported a significant association between plaque burden and recurrent stroke. Kaplan-Meier analysis showed decreased survival with increased plaque burden. Intraplaque hemorrhage and increased enhancement ratio were the other parameters. Studies have reported both characteristics to be associated with a significant risk of recurrence of stroke. Kaplan-Meier analysis also showed an association of increased recurrence of stroke with intraplaque hemorrhage.

DISCUSSION

In the present systematic review, it was observed that recurrent stroke was significantly associated with intraplaque hemorrhage, plaque burden, and increased enhancement ratio, indicating a central role of atherosclerosis and inflammation. Atherosclerosis is the major underlying factor in the formation of plaques, which can lead to stroke. The atherosclerotic process is characterized by elevated plasma low-density lipoprotein (LDL) levels and inflammation of the vessel wall [20]. Wall shear stress is the force exerted by the flowing blood on the vessel wall [21]. It is an important factor in atherosclerosis. Under physiological conditions, parallel flow is protective from the inflammation cascade [22,23]. However, with the deposition of lipids in the subintimal layer and the formation of plaque, blood flow becomes turbulent, wall shear stress changes, and the intima's function is impaired and its integrity compromised [24]. Due to a lack of antioxidants, oxidized LDL (ox-LDL) forms in the subintimal layer, leading to the migration of macrophages and smooth muscle cells [25]. Macrophage-like vascular smooth muscle cells (VSMCs) are also involved in phagocytic clearance of ox-LDL [26]. This results in a necrotic core of the plaque.

The progression of the disease is influenced by plaque erosion, rupture, neovascularization, hemorrhage, and increasing thrombosis. Neovascularization is characterized by immature blood vessels with sparse or absent VSMCs and a lack of tight junctions between endothelial cells, leading to leakage of blood components from the vessels [27]. Hypoxic conditions within the plaque stimulate the secretion of vascular endothelial growth factor (VEGF) and promote the formation of new, leaky blood vessels. These leaky vessels lead to intraplaque hemorrhage [28], especially in the plaque shoulder [29]. Other mechanisms are also hypothesized to lead to intraplaque hemorrhage. The neovessels may undergo eryptosis induced by cholesterol crystals, leading to hemorrhage [30,31]. The content of cholesterol crystals is also an independent predictor of thrombosis [31]. Occasionally, a luminal thrombus rich in erythrocytes may integrate with the plaque, leading to hemorrhage [32]. Luminal blood entry into the plaque is another mechanism leading to hemorrhage [33]. A plaque with an intact fibrous cap has been observed to have fissures, especially those associated with intraplaque hemorrhage [34].

Zhao *et al.* [35] reported that intraplaque hemorrhage was independently associated with a significant increase in lipid core volume. It was also observed in the present systematic review. It has been suggested that intraplaque hemorrhage is associated with the development of plaque vulnerability, which can lead to erosion and rupture. The plaque cap is the single most important component that isolates the thrombogenic core from the bloodstream. It is mostly composed of fibrillary collagens (types I and III) with low thrombogenic potential [36,37]. Therefore, once the fibrin cap is eroded, the core is exposed, which is highly thrombogenic and triggers local thrombus formation, leading to arterial occlusion and stroke. Thus, the intraplaque hemorrhage is significantly associated with recurrent stroke.

Plaque burden was the most extensively studied parameter, showing a significant association with stroke recurrence. Historically, the degree of vessel stenosis was considered the single most important risk factor for recurrent stroke. However, with modern imaging techniques, plaque burden has emerged as an important risk factor for recurrent stroke. Plaque burden includes the volume and composition of the plaque. A higher plaque burden means more plaque surface area, which, in turn, may increase plaque vulnerability. There are several mechanisms by which increased plaque burden

doi: 10.21276/SSR-IIJLS.2026.12.1.35

may lead to recurrent stroke. Higher plaque burden may be associated with a greater proportion of fragile plaque components, particularly in vulnerable plaque, including lipid debris and a thrombotic cap. These components may break off easily, leading to embolism in the territory of the artery and blocking the smaller vessels [38]. Patients with artery-to-artery embolism have also shown a tendency toward increased platelet reactivity [38], leading to greater plaque formation and a greater plaque burden.

When critical stenosis is reached (occlusion >70%), due to a high plaque burden or thrombus formation, there may be significant hypoperfusion in the arterial territory, resulting in impaired clearance of microemboli [39,40]. These result in watershed infarcts. This indirect effect, from decreased clearance of microemboli, is in addition to the direct hypoperfusion caused by arterial stenosis and occlusion. Since the effect is most pronounced at the border areas, it results in watershed infarcts [41,42]. A higher plaque burden is also an indicator of ongoing atherosclerosis and may lead to plaques elsewhere in the body, such as coronary artery disease and aortic arch plaques. These vulnerable plaques in the various arteries are also a potential source of microemboli and may cause recurrent stroke. In patients with stroke, the brain releases pro-inflammatory cytokines. Inflammation plays a crucial role in plaque formation and increased plaque vulnerability [43]. This, in turn, results in recurrent stroke, leading to a vicious cycle. In patients with recurrent stroke, inflammatory processes are intensified, as evidenced by increased enhancement ratios [44].

CONCLUSIONS

The present systematic review shows a significant association of recurrent stroke with plaque burden, intraplaque hemorrhage, and increased enhancement ratio. Vulnerable plaques are an important predictor of stroke recurrence. Therefore, multicenter studies may be conducted to standardize VWI protocols and quantify the risk of recurrent stroke across different plaque characteristics. It is also recommended that clinical scores be devised with VWI biomarkers to predict the risk of recurrence of stroke.

CONTRIBUTION OF AUTHORS

Research concept- Amrit Bansod, Lalit Nirwan
Research design- Amrit Bansod, Harshwardhan Jain, S Agrawal
Supervision- Lalit Nirwan, Harshwardhan Jain
Materials- Amrit Bansod, Lalit Nirwan
Data collection- Tushar Patil, Harshwardhan Jain, S Agrawal
Data analysis and interpretation- Lalit Nirwan, Harshwardhan Jain
Literature search- Tushar Patil, Lalit Nirwan
Writing article- Amrit Bansod, Tushar Patil, Lalit Nirwan, Harshwardhan Jain, S Agrawal
Critical review- Amrit Bansod, Tushar Patil, Lalit Nirwan, Harshwardhan Jain, S Agrawal
Article editing- Amrit Bansod, Tushar Patil, Lalit Nirwan, Harshwardhan Jain, S Agrawal
Final approval- Amrit Bansod, Tushar Patil, Lalit Nirwan, Harshwardhan Jain, S Agrawal

REFERENCES

[1] Pu L, Wang L, Zhang R, Zhao T, Jiang Y, et al. Projected global trends in ischemic stroke incidence, deaths and disability-adjusted life years from 2020 to 2030. Stroke, 2023; 54(5): 1330-39.
[2] Feigin VL, Brainin M, Norrving B, Martins S, Sacco RL, et al. World stroke organization (WSO): Global stroke fact sheet 2022. Int J Stroke, 2022; 17(1): 18-29.
[3] Mohan KM, Wolfe CDA, Rudd AG, Heuschmann PU, Kolominsky-Rabas PL, et al. Risk and cumulative risk of stroke recurrence: a systematic review and meta-analysis. Stroke, 2011; 42(5): 1489-94.
[4] Skajaa N, Adelborg K, Horváth-Puhó E, Rothman KJ, Henderson VW, et al. Risks of stroke recurrence and mortality after first and recurrent strokes in Denmark: A nationwide registry study. Neurol., 2022; 98(4): e329-42.
[5] Yaghi S, Prabhakaran S, Khatri P, Liebeskind DS. Intracranial atherosclerotic disease. Stroke, 2019; 50(5): 1286-93.
[6] Panagiotopoulos E, Stefanou MI, Magoufis G, Safouris A, Kargiotis O, et al. Prevalence, diagnosis and management of intracranial atherosclerosis in White populations: a narrative review. Neurol Res Pract., 2024; 6(1): 54.
[7] Prabhakaran S, Liebeskind DS, Cotsonis G, Nizam A, MYRIAD Investigators, et al. Predictors of early infarct recurrence in patients with symptomatic

intracranial atherosclerotic disease. Stroke, 2021; 52(6): 1961-66.

[8] Zhu B, Liu H, Pan Y, Jing J, CHANCE Investigators, et al. Elevated neutrophil and presence of intracranial artery stenosis increase the risk of recurrent stroke. Stroke, 2018; 49(10): 2294-300.

[9] Wang Y, Liu X, Wu X, Degnan AJ, Malhotra A, et al. Culprit intracranial plaque without substantial stenosis in acute ischemic stroke on vessel wall MRI: A systematic review. Atherosclerosis, 2019; 287: 112-21.

[10]Tian X, Tian B, Shi Z, Wu X, Peng W, et al. Assessment of intracranial atherosclerotic plaques using 3D black-blood MRI: Comparison with 3D time-of-flight MRA and DSA. J Magn Reson Imag., 2021; 53(2): 469-78.

[11]Zhu C, Haraldsson H, Tian B, Meisel K, Ko N, et al. High resolution imaging of the intracranial vessel wall at 3 and 7 T using 3D fast spin echo MRI. MAGMA, 2016; 29(3): 559-70.

[12]Mandell DM, Mossa-Basha M, Qiao Y, Hess CP, Hui F, et al. Intracranial Vessel Wall MRI: Principles and Expert Consensus Recommendations of the American Society of Neuroradiology. AJNR Am J Neuroradiol., 2017; 38(2): 218-29. doi: 10.3174/ajnr.A4893.

[13]Lu M, Peng P, Cui Y, Qiao H, Li D, et al. Association of progression of carotid artery wall volume and recurrent transient ischemic attack or stroke: A magnetic resonance imaging study. Stroke, 2018; 49(3): 614-20.

[14]Ran Y, Wang Y, Zhu M, Wu X, Malhotra A, et al. Higher plaque burden of middle cerebral artery is associated with recurrent ischemic stroke: A quantitative magnetic resonance imaging study. Stroke, 2020; 51(2): 659-62.

[15]Shi Z, Li J, Zhao M, Zhang X, Degnan AJ, et al. Progression of plaque burden of intracranial atherosclerotic plaque predicts recurrent stroke/transient ischemic attack: A pilot follow-up study using higher-resolution MRI. J Magn Reson Imaging, 2021; 54(2): 560-70.

[16]Shen ZZ, Ren SJ, Wu RR, Su CQ, Ge S, et al. Temporal changes in plaque characteristics after treatment and their relationship with stroke recurrence: A quantitative study using magnetic resonance imaging. Quant Imaging Med Surg., 2022; 12(9): 4559-69.

[17]van Dam-Nolen DHK, Truijman MTB, van der Kolk AG, Liem MI, PARISK Study Group, et al. Carotid plaque characteristics predict recurrent ischemic stroke and TIA: The PARISK (Plaque at risk) study. JACC Cardiovasc Imaging, 2022; 15(10): 1715-26.

[18]Lv Y, Ma X, Zhao W, Ju J, Yan P, et al. 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., 2024; 34(5): 3022-31.

[19]Yao W, Chen H, Huang K, Peng W, Zhang X, et al. Atherosclerotic plaque evolution predicts cerebral ischemic events in patients with intracranial atherosclerosis: a multicentre longitudinal study using high-resolution MRI. Eur Radiol., 2025; 35(6): 3238-48.

[20]Zhu Y, Xian X, Wang Z, Bi Y, Chen Q, et al. Research progress on the relationship between atherosclerosis and inflammation. Biomol., 2018; 8(3): 80.

[21]Malik J, Novakova L, Valerianova A, Chytilova E, Lejsek V, et al. Wall shear stress alteration: A local risk factor of atherosclerosis. Curr Atheroscler Rep., 2022; 24(3): 143-51.

[22]Humphrey JD, Schwartz MA. Vascular Mechanobiology: Homeostasis, adaptation, and disease. Annu Rev Biomed Eng., 2021; 23: 1-27.

[23]Sluiter TJ, van Buul JD, Huveneers S, Quax PH, de Vries MR. Endothelial barrier function and leukocyte transmigration in atherosclerosis. Biomed., 2021; 9(4): 328.

[24]Zhou M, Yu Y, Chen R, Liu X, Hu Y, et al. Wall shear stress and its role in atherosclerosis. Front Cardiovasc Med., 2023; 10: 1083547.

[25]Khatana C, Saini NK, Chakrabarti S, Saini V, Sharma A, et al. Mechanistic insights into the oxidized low-density lipoprotein-induced atherosclerosis. Oxid Med Cell Longev., 2020; 2020: 5245308.

[26]Grootaert MO, Bennett MR. Vascular smooth muscle cells in atherosclerosis: Time for a re-assessment. Cardiovasc Res., 2021; 117(11): 2326-39.

[27]Yoshida K, Yang T, Yamamoto Y, Kurosaki Y, Funaki T, et al. Expansive carotid artery remodeling: Possible marker of vulnerable plaque. J Neurosurg., 2019; 133(5): 1435-40.

doi: 10.21276/SSR-IIJLS.2026.12.1.35

[28]Nardi V, Benson C, Bois MC, Saba L, Larson AS, et al. Carotid plaques from symptomatic patients with mild stenosis is associated with intraplaque hemorrhage. Hypertens., 2022; 79(1): 271-82.

[29]Jeziorska M, Woolley DE. Local neovascularization and cellular composition within vulnerable regions of atherosclerotic plaques of human carotid arteries. J Pathol., 1999; 188: 189-96.

[30]Abela GS, Aziz K. Cholesterol crystals rupture biological membranes and human plaques during acute cardiovascular events- A novel insight into plaque rupture by scanning electron microscopy. Scanning, 2006; 28: 1-10.

[31]Abela GS, Aziz K, Vedre A, Pathak DR, Talbott JD, et al. Effect of cholesterol crystals on plaques and intima in arteries of patients with acute coronary and cerebrovascular syndromes. Am J Cardiol., 2009; 103(7): 959-68.

[32]Michel JB, Martin-Ventura JL, Nicoletti A, et al. Pathology of human plaque vulnerability: Mechanisms and consequences of intraplaque haemorrhages. Atherosclerosis, 2014; 234(2): 311-19.

[33]Virmani R, Kolodgie FD, Burke AP, Finn AV, Gold HK, et al. Atherosclerotic plaque progression and vulnerability to rupture: Angiogenesis as a source of intraplaque hemorrhage. Arterioscler Thromb Vasc Biol., 2005; 25(10): 2054-61.

[34]Daemen MJ, Ferguson MS, Gijsen FJ, Hippe DS, Kooi ME, et al. Carotid plaque fissure: An underestimated source of intraplaque hemorrhage. Atherosclerosis, 2016; 254: 102-08.

[35]Zhao XQ, Sun J, Hippe DS, Isquith DA, AIM-HIGH Carotid MRI Substudy Investigators, et al. Magnetic resonance imaging of intraplaque hemorrhage and plaque lipid content with continued lipid-lowering therapy: Results of a magnetic resonance imaging substudy in AIM-HIGH. Circ Cardiovasc Imag., 2022; 15(11): e014229.

[36]Barter PJ. Inhibition of endothelial cell adhesion molecule expression by high-density lipoproteins. Clin Exp Pharmacol Physiol., 1997; 24(3–4): 286–87. doi: 10.1111/j.1440-1681.1997.tb01821.x.

[37]Farb A, Burke AP, Tang AL, Liang TY, Mannan P, et al. Coronary plaque erosion without rupture into a lipid core. A frequent cause of coronary thrombosis in sudden coronary death. Circulation, 1996; 93(7): 1354–63.

[38]Ha SH, Chang JY, Lee SH, Lee KM, Heo SH, et al. Mechanism of stroke according to the severity and location of atherosclerotic middle cerebral artery disease. J Stroke Cerebrovasc Dis., 2021; 30(2): 105503.

[39]Chatzikonstantinou A, Wolf ME, Schaefer A, Hennerici MG. Asymptomatic and symptomatic carotid stenosis: An obsolete classification?? Stroke Res Treat., 2012; 2012: 340798. doi: 10.1155/2012/340798.

[40]Archie JP. Critical arterial stenosis: An enigma explained. Eur J Vasc Endovasc Surg., 2023; 65(3): 457-59.

[41]Mangla R, Kolar B, Almast J, Ekholm SE. Border zone infarcts: Pathophysiologic and imaging characteristics. Radiographics, 2011; 31(5): 1201-14. doi: 10.1148/rg.315105014.

[42]ElSadek A, Gaber A, Afifi H, Farag S, Salaheldien N. Microemboli versus hypoperfusion as an etiology of acute ischemic stroke in Egyptian patients with watershed zone infarction. Egypt J Neurol Psychiatr Neurosurg., 2019; 55(1): 2.

[43]Hansson GK, Libby P, Tabas I. Inflammation and plaque vulnerability. J Intern Med., 2015; 278(5): 483-93.

[44]Hur J, Park J, Kim YJ, Lee HJ, Shim HS, et al. Use of contrast enhancement and high-resolution 3D black-blood MRI to identify inflammation in atherosclerosis. JACC Cardiovasc Imaging, 2010; 3(11): 1127-35.