Section: General Medicine

Original Research Article

UTILITY OF MONOCYTE TO HIGH-DENSITY LIPOPROTEIN CHOLESTEROL RATIO IN ASSESSMENT OF SEVERITY AND PROGNOSIS IN ACUTE ISCHEMIC STROKE: A PROSPECTIVE OBSERVATIONAL STUDY FROM A SOUTH INDIAN TERTIARY CARE CENTRE

Duggireddy Rakesh Kumar Reddy[1], Sudha V[2], Uma M A[3], Siri Chandana Putta[4], Amarnath Reddy[5]

[1]Postgraduate Final Year, Department of General Medicine, PES Institute of Medical Sciences and Research, Kuppam, India.
[2]Assistant Professor, Department of General Medicine, PES Institute of Medical Sciences and Research, Kuppam, India.
[3]Professor and Head of the Department, Department of General Medicine, PES Institute of Medical Sciences and Research, Kuppam, India.
[4]Postgraduate Final Year, Department of General Medicine, PES Institute of Medical Sciences and Research, Kuppam, India.
[5]Postgraduate Final Year, Department of General Medicine, PES Institute of Medical Sciences and Research, Kuppam, India.

Received *: 12/05/2026*
Received in revised form *: 30/06/2026*
Accepted *: 14/07/2026*

Corresponding Author:
Dr. Duggireddy Rakesh Kumar Reddy,
Postgraduate Final Year, Department of General Medicine, PES Institute of Medical Sciences and Research, Kuppam, India.
Email: drkreddy241298@gmail.com

DOI: 10.70034/ijmedph.2026.3.172

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 1074-1079

ABSTRACT

Background: Acute ischemic stroke is a common neurological emergency with substantial morbidity, and inflammatory-lipid markers such as the monocyte-to-high-density lipoprotein cholesterol ratio (MHR) have been proposed as simple indicators of disease severity. **Objectives:** To estimate the ratio of monocytes to HDL cholesterol in acute ischemic stroke patients and to correlate the monocyte-to-HDL cholesterol ratio with the National Institutes of Health Stroke Scale (NIHSS) score for assessing stroke severity.

Materials and Methods: A hospital-based prospective observational study was conducted over 18 months in the Department of General Medicine at a tertiary care centre in Kuppam, Andhra Pradesh. Consecutive patients diagnosed with acute ischemic stroke within 24 hours of symptom onset were enrolled by purposive sampling after written informed consent (n=100). Clinical assessment included Glasgow Coma Scale and NIHSS scoring at admission and before discharge, with diagnosis confirmed by CT or MRI brain imaging. Peripheral venous blood was collected at admission; complete blood count and fasting lipid profile were performed and MHR was calculated. Data were analysed in SPSS v23.0; Pearson correlation assessed associations between monocyte measures, HDL, MHR, and NIHSS, with $p<0.05$ considered significant.

Results: Among 100 participants, the majority were aged 60–69 years (38.0%) or ≥70 years (31.0%), with a nearly equal gender distribution (male 51.0%, female 49.0%). Diabetes mellitus and hypertension were present in 35.0% and 34.0% respectively, while smoking and alcohol consumption were reported in 30.0% and 27.0%. Mean systolic and diastolic blood pressures were 147.32±22.89 mmHg and 83.72±11.71 mmHg. Mean monocyte percentage was 6.33±1.81, mean HDL cholesterol was 35.48±11.71 mg/dL, mean MHR was 0.20±0.09, and mean NIHSS score was 7.42±2.86. Monocyte percentage showed a significant positive correlation with NIHSS score ($r=0.339$, $p=0.0006$), whereas HDL cholesterol showed a weak negative non-significant correlation ($r=-0.074$, $p=0.4636$). MHR also demonstrated a significant positive correlation with NIHSS score ($r=0.227$, $p=0.0229$).

Conclusion: Monocyte-to-HDL ratio and monocyte percentage were significantly associated with stroke severity in acute ischemic stroke patients. These findings suggest that MHR may serve as a simple, inexpensive, and

readily available biomarker for early assessment of stroke severity and prognostication in acute ischemic stroke.
Keywords: Acute ischemic stroke; monocyte-to-HDL ratio; NIHSS; inflammation; dyslipidemia; stroke severity; prognosis.

INTRODUCTION

Stroke is a leading cause of mortality and disability worldwide, defined as an abrupt onset of neurological deficit attributable to vascular causes, primarily ischemic or haemorrhagic events.[1] Ischemic stroke constitutes nearly 70–80% of all cases globally and arises from obstruction of cerebral blood flow due to thrombosis, embolism, or arterial stenosis.[2] The global burden is substantial, with ischemic stroke ranking among the top causes of death and disability-adjusted life years worldwide, and the lifetime risk of stroke in adults aged 25 years and above estimated at one in four globally.[3,4]

India bears a disproportionately high burden of stroke, reflecting demographic transition and rising prevalence of hypertension, diabetes, dyslipidaemia, and lifestyle change, with crude prevalence estimated at 84 to 262 per 100,000 population and an annual incidence of 105 to 152 per 100,000.[5,6] Stroke mortality in India is significantly higher than in developed countries, reflecting challenges in acute management, poor awareness, and delayed access to specialised care, and recent data suggest an increasing trend of ischemic stroke among younger populations, carrying substantial socioeconomic implications.[7,8]

The pathophysiology of ischemic stroke involves excitotoxicity, oxidative stress, endothelial dysfunction, and immune-inflammatory responses, with inflammation recognised as a critical determinant not only in the initiation of atherosclerosis but also in plaque destabilisation and progression of cerebral ischaemia.[9,10] Monocytes, as key mediators of innate immunity, contribute to endothelial injury and plaque rupture leading to thromboembolic events, whereas high-density lipoprotein cholesterol (HDL-C) exerts anti-inflammatory and antioxidant effects that stabilise the vascular endothelium and modulate immune activation.[11,12]

The monocyte-to-HDL cholesterol ratio (MHR) has emerged as a composite biomarker reflecting the balance between pro-inflammatory and anti-inflammatory forces within the vascular system, and elevated MHR has been associated with worse outcomes in cardiovascular diseases including coronary artery disease and acute coronary syndromes.[13,14] Since monocyte activation and HDL dysfunction are central to ischaemic pathophysiology, MHR may provide a cost-effective, readily available, and biologically meaningful marker to stratify stroke patients at admission.

Despite advances in neuroimaging and laboratory biomarkers, early prediction of stroke severity and outcomes remains a challenge in resource-constrained settings such as India. The NIHSS is widely used for clinical assessment but does not incorporate molecular or cellular biomarkers that may reflect underlying disease biology. In this context, MHR offers novelty as an accessible biomarker that could complement clinical scales; however, most existing data come from cardiovascular literature, and studies specifically evaluating MHR in acute ischemic stroke remain limited. Given the rising burden of ischemic stroke worldwide and in India, coupled with the need for inexpensive and reliable biomarkers, this study was undertaken to investigate the prognostic value of MHR in acute ischemic stroke.

Aim and Objectives

Objectives

1. To estimate the ratio of monocytes to HDL cholesterol in acute ischemic stroke patients.
2. To correlate the monocyte-to-high-density lipoprotein cholesterol ratio with the NIHSS score for assessing stroke severity.

MATERIALS AND METHODS

This hospital-based prospective observational study was conducted in the Department of General Medicine at a tertiary care teaching hospital in Kuppam, Andhra Pradesh, India, over 18 months. Both male and female patients aged 18 to 70 years admitted with acute ischemic stroke within 24 hours of symptom onset were included. Patients with haemorrhagic stroke, haematological malignancy, venous sinus thrombosis, autoimmune disease, hepatic or renal disorders, connective tissue disorders, moribund conditions, seizure disorders, mental or physical illness affecting assessment, and previous history of stroke were excluded.

The minimum sample size was calculated using the formula for estimation of a single proportion ($n = 4pq/d^2$), assuming an anticipated proportion of 50% and an absolute precision of 10%, yielding a minimum requirement of 100 patients; all 100 were enrolled by purposive (consecutive) sampling after written informed consent.

After institutional ethics committee approval, a structured proforma was used to record demographic details, medical history, and clinical examination findings. Neurological assessment included Glasgow Coma Scale, cranial nerve examination, sensory and motor evaluation, and NIHSS scoring, with stroke severity classified into minor, moderate, moderate-to-severe, and severe categories. CT or MRI brain imaging confirmed the

diagnosis. Peripheral venous blood samples were collected under aseptic precautions at admission and again before discharge; investigations included complete blood count, fasting lipid profile, coagulation profile, and liver and renal function tests, from which MHR was calculated. Confidentiality was maintained using unique identification numbers, and personal identifiers were not disclosed.

Data were entered in Microsoft Excel and analysed using SPSS version 23.0 (SPSS Inc., Chicago, IL, USA). Categorical variables were expressed as frequencies and percentages and compared using the Chi-square or Fisher's Exact test; continuous variables were expressed as mean±SD and compared using Student's t-test or the Mann-Whitney U test depending on distribution. Correlations between MHR, monocyte percentage, HDL, and NIHSS score were assessed using Pearson correlation coefficients, and receiver operating characteristic curve analysis was used to evaluate the predictive accuracy of MHR for stroke severity. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 100 patients with acute ischemic stroke were enrolled. The age distribution demonstrated a predominance of older adults: the 60–69 years group constituted the largest proportion (38.0%), followed by patients aged ≥70 years (31.0%), 51–59 years (18.0%), 30–39 years (11.0%), and 41–49 years (2.0%). Gender distribution was nearly balanced, with males comprising 51.0% and females 49.0%. Diabetes mellitus was present in 35.0% of participants and hypertension in 34.0%, while smoking history was reported by 30.0% and alcohol consumption by 27.0%. Mean systolic and diastolic blood pressures at admission were 147.32±22.89 mmHg and 83.72±11.71 mmHg respectively. Mean monocyte percentage was 6.33±1.81, mean HDL cholesterol was 35.48±11.71 mg/dL, mean MHR was 0.20±0.09, and mean NIHSS score was 7.42±2.86. These baseline characteristics are summarised in Table 1.

Table 1: Baseline demographic, clinical, and biochemical characteristics of the study population (n=100)

Variable	n / Mean	% / SD
Age 30–39 years, n (%)	11	11.0%
Age 41–49 years, n (%)	2	2.0%
Age 51–59 years, n (%)	18	18.0%
Age 60–69 years, n (%)	38	38.0%
Age ≥70 years, n (%)	31	31.0%
Male sex, n (%)	51	51.0%
Female sex, n (%)	49	49.0%
Diabetes mellitus, n (%)	35	35.0%
Hypertension, n (%)	34	34.0%
Smoking, n (%)	30	30.0%
Alcohol consumption, n (%)	27	27.0%
Mean systolic BP (mmHg)	147.32	±22.89
Mean diastolic BP (mmHg)	83.72	±11.71
Mean monocyte (%)	6.33	±1.81
Mean HDL cholesterol (mg/dL)	35.48	±11.71
Mean monocyte-to-HDL ratio (MHR)	0.20	±0.09
Mean NIHSS score	7.42	±2.86

BP, blood pressure; HDL, high-density lipoprotein; MHR, monocyte-to-HDL ratio; NIHSS, National Institutes of Health Stroke Scale.

Radiological assessment demonstrated that anterior circulation strokes were the most common pattern, with middle cerebral artery (MCA) territory/anterior circulation infarcts accounting for 49.0% of cases, followed by posterior circulation infarcts (18.0%), posterior cerebral artery (PCA) territory infarcts (14.0%), anterior cerebral artery (ACA) territory infarcts (7.0%), multifocal/multiple territory infarcts (7.0%), small vessel/lacunar infarcts (4.0%), and non-specific chronic ischaemic changes (1.0%), as summarised in Table 2.

Table 2: Distribution of CT/MRI brain findings by stroke territory (n=100)

Stroke territory	n	%
Middle cerebral artery (MCA)/anterior circulation infarct	49	49.0%
Posterior circulation infarct (brainstem/cerebellum/thalamic)	18	18.0%
Posterior cerebral artery (PCA) territory infarct	14	14.0%
Anterior cerebral artery (ACA) territory infarct	7	7.0%
Multifocal / multiple territory infarcts	7	7.0%
Small vessel / lacunar infarcts	4	4.0%
Non-specific / chronic ischaemic changes	1	1.0%
Total	100	100.0%

Systolic blood pressure categories showed a statistically significant association with stroke severity (p=0.035): among patients with SBP <160 mmHg, 26.56% had minor and 73.44% had moderate stroke, compared with 6.45% minor and 93.55% moderate in the 160–180 mmHg category, and 100% moderate stroke in those with SBP >180 mmHg. Diastolic blood pressure categories similarly showed a significant association (p=0.004), with 30.61% minor and 69.39% moderate stroke among patients with DBP <90 mmHg, compared with 7.84% minor and 92.16% moderate in the 90–110 mmHg category. HDL categories were significantly associated with stroke severity (p=0.019): low HDL was associated with 84.62% moderate stroke, normal HDL with 77.42% moderate stroke, and high HDL with an equal 50:50 split between minor and moderate stroke. Monocyte categories also showed a significant association with severity (p=0.011), with the normal monocyte category showing the highest proportion of moderate stroke (84.81%). However, monocyte categories were not significantly associated with HDL categories (p=0.777), indicating that although both variables independently correlated with stroke severity, they were not directly associated with each other in categorical analysis. These associations are detailed in Table 3.

Table 3: Association of clinical and biochemical categories with stroke severity (n=100)

Category	Sub-group	Minor stroke n (%)	Moderate stroke n (%)	p-value
Systolic BP (mmHg)	<160	17 (26.56%)	47 (73.44%)	0.035*
	160–180	2 (6.45%)	29 (93.55%)	
	>180	0 (0.00%)	5 (100.00%)	
Diastolic BP (mmHg)	<90	15 (30.61%)	34 (69.39%)	0.004*
	90–110	4 (7.84%)	47 (92.16%)	
HDL category	Low	10 (15.38%)	55 (84.62%)	0.019*
	Normal	7 (22.58%)	24 (77.42%)	
	High	2 (50.00%)	2 (50.00%)	
Monocyte category	Low	2 (50.00%)	2 (50.00%)	0.011*
	Normal	12 (15.19%)	67 (84.81%)	
	High	5 (29.41%)	12 (70.59%)	

**p<0.05, Chi-square/Fisher's Exact test. Association between monocyte categories and HDL categories was not statistically significant (low monocyte: 50.0% low HDL, 50.0% normal HDL; normal monocyte: 64.56% low HDL, 30.38% normal HDL; high monocyte: 70.59% low HDL, 29.41% normal HDL; p=0.777). BP, blood pressure; HDL, high-density lipoprotein.*

Monocyte percentage had a mean of 6.33 (SD 1.81) and demonstrated a statistically significant positive correlation with NIHSS score (r=0.339, p=0.0006), indicating that higher monocyte levels were associated with greater neurological deficit. HDL cholesterol, with a mean of 35.48 mg/dL (SD 11.71), showed a weak negative correlation with NIHSS that did not reach statistical significance (r=−0.074, p=0.4636). The monocyte-to-HDL ratio, with a mean of 0.20 (SD 0.09), demonstrated a statistically significant positive correlation with NIHSS score (r=0.227, p=0.0229), confirming that higher MHR values were associated with greater stroke severity. These correlations are presented in Table 4.

Table 4: Correlation between monocyte percentage, HDL cholesterol, MHR, and NIHSS score (n=100)

Parameter	Mean	SD	Correlation coefficient (r) with NIHSS	p-value
Monocyte (%)	6.33	1.81	0.339	0.0006*
HDL cholesterol (mg/dL)	35.48	11.71	−0.074	0.4636
Monocyte-to-HDL ratio (MHR)	0.20	0.09	0.227	0.0229*
NIHSS score (reference)	7.42	2.86	—	—

**p<0.05, Pearson correlation. NIHSS, National Institutes of Health Stroke Scale; SD, standard deviation.*

DISCUSSION

Acute ischemic stroke remains a major cause of mortality and long-term disability worldwide, and early identification of markers reflecting stroke severity is important for prompt prognostication and risk stratification. The present study demonstrates that monocyte percentage and MHR are significantly associated with stroke severity, whereas HDL cholesterol alone does not show a significant independent correlation with NIHSS score. These findings suggest that composite inflammatory-lipid markers may better reflect the underlying pathophysiological burden of acute ischemic stroke than isolated lipid parameters, supporting the concept that ischemic stroke is not merely a vascular occlusive event but a complex thrombo-inflammatory process involving systemic immune activation, endothelial dysfunction, and neuroinflammation.

The age distribution of the study population, with a majority of patients older than 60 years, is consistent with the established epidemiology of stroke, as advancing age is associated with cumulative

vascular risk exposure, progressive endothelial dysfunction, and impaired cerebrovascular autoregulation. The nearly equal gender distribution indicates that the observed associations between inflammatory markers and stroke severity are unlikely to be significantly confounded by sex imbalance, while the substantial proportion of participants with diabetes mellitus, hypertension, smoking, and alcohol use emphasises the persistent contribution of modifiable cardiometabolic risk factors to the pathogenesis of ischemic stroke.

Radiological assessment demonstrated that anterior circulation strokes, particularly MCA territory infarcts, constituted the predominant stroke subtype in this cohort, consistent with the known predominance of anterior circulation infarcts in hospitalised acute ischemic stroke populations. Because infarct location and vascular territory significantly influence NIHSS score, lesion heterogeneity must be considered when interpreting biomarker correlations; nevertheless, the observed relationship between inflammatory markers and NIHSS across diverse infarct patterns suggests that these biomarkers may reflect systemic stroke burden beyond anatomical lesion location alone.

A significant association was observed between elevated systolic and diastolic blood pressure categories and greater stroke severity. Higher blood pressure at admission may represent a combination of pre-existing hypertension, physiological stress response, sympathetic activation, and impaired cerebral autoregulation. Since systemic stress responses may also influence leukocyte dynamics, blood pressure and inflammatory biomarkers should be interpreted together when assessing acute stroke severity, as their coexistence in more severe stroke may reflect the integrated physiological response to larger infarct burden and heightened systemic stress.

Monocyte percentage demonstrated a moderate positive correlation with NIHSS score, indicating that higher monocyte levels were associated with greater neurological deficit. Monocytes play a central role in the inflammatory cascade following cerebral ischaemia through endothelial adhesion, transmigration into ischaemic brain tissue, cytokine release, oxidative stress generation, and promotion of blood-brain barrier disruption.[11,15] Increased peripheral monocyte count may therefore reflect enhanced systemic inflammatory activation accompanying larger infarct burden and more severe neurological injury, consistent with associations between monocyte-related inflammatory markers and stroke severity reported in previous studies.[16,17]

Although monocyte percentage correlated significantly with NIHSS, HDL cholesterol alone did not demonstrate a significant relationship with stroke severity. This may be explained by the fact that HDL concentration does not necessarily reflect HDL functionality during acute inflammatory states; in acute ischemic stroke, HDL particles may become dysfunctional, losing their antioxidant and anti-inflammatory properties despite preserved circulating concentration, while acute phase responses can transiently alter lipid profiles, reducing the prognostic relevance of isolated HDL measurement during the acute stage of stroke.

In contrast, MHR demonstrated a statistically significant positive correlation with NIHSS score, supporting its potential value as a composite biomarker of stroke severity. MHR integrates two biologically opposing mechanisms, monocyte-mediated inflammation and HDL-mediated vascular protection, and a higher MHR may therefore reflect increased inflammatory burden, reduced anti-inflammatory lipid protection, or both. This composite nature likely explains its association with stroke severity compared with HDL alone, consistent with cardiovascular literature in which MHR has been associated with adverse outcomes in coronary artery disease and other atherosclerotic disorders.[14,18] The positive correlation between MHR and stroke severity observed in the present study is consistent with emerging stroke-specific literature evaluating leukocyte-derived inflammatory indices as prognostic markers.[17,19]

The magnitude of correlation between MHR and NIHSS, although statistically significant, was modest, indicating that MHR should not be considered a standalone prognostic tool. Stroke severity is multifactorial and influenced by infarct volume, lesion location, collateral circulation, time to presentation, reperfusion status, and baseline comorbidities; MHR is therefore best interpreted as an adjunctive biomarker that complements established clinical and radiological assessment rather than replacing it. Its practical value lies in the fact that it can be derived from routinely available laboratory investigations without additional cost or specialised testing, which is particularly relevant in resource-limited settings such as many Indian tertiary care hospitals, where automated reporting of MHR alongside complete blood count and lipid profile could facilitate rapid identification of patients at higher risk of severe neurological deficit.

Certain limitations of this analysis must be considered when interpreting the observed associations, since the cross-sectional correlation design precludes causal inference, and elevated monocyte counts or MHR may represent markers of greater tissue injury rather than direct mediators of stroke severity.

Limitations

This study has certain limitations. It was conducted at a single tertiary care centre with a relatively modest sample size, which may limit generalisability and reduce the power to detect weaker associations. Stroke subtype classification based on aetiology was not performed, preventing evaluation of whether MHR varies across different ischemic stroke mechanisms, and serial dynamic changes in MHR during hospitalisation were not analysed, which may provide additional prognostic information beyond baseline measurement. Long-term functional outcomes were not assessed, as

follow-up was limited to the hospital stay, and stroke severity itself is multifactorial, influenced by infarct volume, lesion location, collateral circulation, and time to presentation, all of which may confound the observed biomarker associations.

CONCLUSION

The present study demonstrates that inflammatory biomarkers, particularly monocyte percentage and monocyte-to-high-density lipoprotein ratio, are significantly associated with stroke severity in patients with acute ischemic stroke. The positive correlation of these markers with NIHSS score suggests that increased inflammatory activity at admission is associated with greater neurological impairment and more severe clinical presentation, whereas HDL cholesterol alone did not demonstrate a significant independent association, indicating that combined inflammatory-lipid indices may provide superior prognostic information compared with isolated lipid parameters.

These findings support the utility of the monocyte-to-HDL ratio as a simple, inexpensive, and readily available biomarker for assessing stroke severity and early prognosis in acute ischemic stroke. Incorporation of this ratio into routine clinical evaluation may enhance early risk stratification and prognostic assessment, assisting clinicians in identifying patients at greater risk of severe neurological deficits. Further large-scale multicentre prospective studies are warranted to validate its prognostic utility, determine standardised cutoff values, and establish its incremental value when integrated into multivariable stroke prediction models.

REFERENCES

1. Sacco RL, Kasner SE, Broderick JP, et al. An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke. 2013;44(7):2064–2089.
2. Feske SK. Ischemic stroke. Am J Med. 2021;134(12):1457–1464.
3. Li XY, Kong XM, Yang CH, et al. Global, regional, and national burden of ischemic stroke, 1990–2021: an analysis of data from the global burden of disease study 2021. EClinicalMedicine. 2024;75:102758.
4. NCD Countdown 2030 Collaborators. NCD countdown 2030: worldwide trends in non-communicable disease mortality and progress towards sustainable development goal target 3.4. Lancet. 2018;392(10152):1072–1088.
5. Pandian JD, Sudhan P. Stroke epidemiology and stroke care services in India. J Stroke. 2013;15(3):128–134.
6. Kamalakannan S, Gudlavalleti ASV, Gudlavalleti VSM, et al. Incidence and prevalence of stroke in India: a systematic review. Indian J Med Res. 2017;146(2):175–185.
7. Sridharan SE, Unnikrishnan JP, Sukumaran S, et al. Incidence, types, risk factors, and outcome of stroke in a developing country: the Trivandrum stroke registry. Stroke. 2009;40(4):1212–1218.
8. Das SK, Banerjee TK. Stroke: Indian scenario. Circulation. 2008;118(25):2719–2724.
9. Endres M, Moro MA, Nolte CH, et al. Immune pathways in etiology, acute phase, and chronic sequelae of ischemic stroke. Circ Res. 2022;130(8):1167–1186.
10. Libby P, Ridker PM, Hansson GK. Inflammation in atherosclerosis: from pathophysiology to practice. J Am Coll Cardiol. 2009;54(23):2129–2138.
11. Jin R, Yang G, Li G. Inflammatory mechanisms in ischemic stroke: role of inflammatory cells. J Leukoc Biol. 2010;87(5):779–789.
12. Reina SA, Llabre MM, Allison MA, et al. HDL cholesterol and stroke risk: the multi-ethnic study of atherosclerosis. Atherosclerosis. 2015;243(1):314–319.
13. Caimi G, Lo Presti R, Urso C, et al. Neutrophil/HDL-C, lymphocyte/HDL-C and monocyte/HDL-C in subjects with asymptomatic carotid atherosclerosis. Clin Hemorheol Microcirc. 2024;88(1):1–11.
14. Cetin MS, Ozcan Cetin EH, Kalender E, et al. Monocyte to HDL cholesterol ratio predicts coronary artery disease severity and future major cardiovascular adverse events in acute coronary syndrome. Heart Lung Circ. 2016;25(11):1077–1086.
15. Shi K, Tian DC, Li ZG, et al. Global brain inflammation in stroke. Lancet Neurol. 2019;18(11):1058–1066.
16. Liberale L, Montecucco F, Bonaventura A, et al. Monocyte count at onset predicts poststroke outcomes during a 90-day follow-up. Eur J Clin Invest. 2017;47(9):702–710.
17. Gkantzios A, Tsiptsios D, Karapepera V, et al. Monocyte to HDL and neutrophil to HDL ratios as potential ischemic stroke prognostic biomarkers. Neurol Int. 2023;15(1):301–317.
18. Ganjali S, Gotto AM Jr, Ruscica M, et al. Monocyte-to-HDL-cholesterol ratio as a prognostic marker in cardiovascular diseases. J Cell Physiol. 2018;233(12):9237–9246.
19. Cao X, Zhu Q, Xia X, et al. The correlation between novel peripheral blood cell ratios and 90-day mortality in patients with acute ischemic stroke. PLoS One. 2020;15(8):e0238312.