Medicinay Laboratorio
Online ISSN- 2500-7106| Print ISSN- 0123-2576
Available online on: https://medicinaylaboratorio.com/

Original Article

The Importance of Monocyte-to-High-Density Lipoprotein Cholesterol Ratio in Predicting Stroke Severity and In-Hospital Outcome in Acute Ischemic Stroke

Dr. L. Ashwini[1], Dr. Krishnamurthy H.A.[2], Dr. Sharanprasad Kolar[3], Dr. Suchitra V. Naganagoudar[4]

[1]Postgraduate, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India.
[2]Professor, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India.
[3]Postgraduate, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India.
[4]Postgraduate, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India.

OPEN ACCESS

Corresponding Author:

Dr. L. Ashwini
Postgraduate, Department of General Medicine, Mysore Medical College and Research Institute, Mysore, Karnataka, India.

Received: 10-04-2026
Revised: 02-05-2026
Accepted: 18-06-2026
Available online: 29-06-2026

ABSTRACT

Background: Globally, acute ischaemic stroke (AIS) is a leading cause of death and chronic disability. Lipid metabolism and inflammation are crucial to the development and progression of AIS. A new inflammatory biomarker that reflects the equilibrium between pro-inflammatory monocytes and the beneficial anti-inflammatory effects of HDL cholesterol is the Monocyte-to-High-Density Lipoprotein Cholesterol Ratio (MHR). The purpose of this study was to assess the importance of MHR in predicting in-hospital outcomes and stroke severity in patients with AIS.**Methods:** A prospective observational study was conducted in the Department of General Medicine, K.R. Hospital, Mysore Medical College and Research Institute, Mysuru, from April 2024 to September 2025. Sixty-three patients aged 18–80 years with clinically and radiologically confirmed acute ischemic stroke were included. Clinical details, risk factors, laboratory investigations, and neuroimaging findings were recorded. MHR was calculated using admission monocyte count and HDL cholesterol levels. The National Institutes of Health Stroke Scale (NIHSS) was used to measure the severity of the stroke, and complications, length of hospital stay, and clinical outcomes were used to evaluate in-hospital outcomes. SPSS was used for statistical analysis, and $p < 0.05$ was deemed significant. **Results:** The majority of patients were aged 40–50 years (34.9%), with a slight male predominance (53.9%). Diabetes mellitus (61.9%) and hypertension (46.0%) were common comorbidities. Higher MHR values were significantly associated with aspiration pneumonia and sepsis ($p<0.0001$). Patients with mortality and residual neurological deficits demonstrated significantly higher MHR values compared to those who improved clinically ($p<0.0001$). MHR showed a strong positive correlation with hospital stay duration ($r=0.820$, $p<0.0001$) and NIHSS score on day 7 ($r=0.717$, $p<0.0001$), indicating greater neurological severity and poorer prognosis.**Conclusion**MHR is a simple, inexpensive, and readily available biomarker that correlates significantly with stroke severity, in-hospital complications, prolonged hospitalization, and adverse clinical outcomes in acute ischemic stroke. Incorporation of MHR into routine clinical assessment may facilitate early risk stratification and improve patient management.

***Keywords**: Acute Ischemic Stroke, Monocyte-to-HDL Cholesterol Ratio, MHR, Inflammation, NIHSS, Stroke Severity, Prognostic Biomarker, In-Hospital Outcome.*

INTRODUCTION

Despite considerable advancements in neuroimaging, reperfusion treatments, and organised stroke care, acute ischaemic stroke (AIS) continues to be a leading cause of death and long-term disability globally. Clinical outcomes after AIS vary considerably, ranging from complete recovery to severe neurological impairment or death. This variability is influenced not only by the extent of cerebral ischemia and timeliness of treatment but also by the patient's underlying inflammatory and metabolic status. Increasing evidence suggests that stroke is not merely a focal vascular event but a systemic inflammatory disorder in which immune and lipid-related factors significantly affect disease progression and recovery.[1,2]

The two main causes of ischaemic stroke, atherosclerosis and thromboembolism, are both significantly influenced by inflammation. Monocytes are key inflammatory cells that contribute to endothelial dysfunction, plaque instability, thrombus formation, and blood–brain barrier disruption through the release of cytokines and reactive oxygen species. Larger infarct sizes, early neurological decline, and worse functional outcomes after stroke have all been linked to elevated monocyte numbers.[2,3]
High-density lipoprotein cholesterol (HDL-C), on the other hand, has anti-inflammatory, antioxidant, antithrombotic, and endothelial-protective properties. In addition to its function in reverse cholesterol transport, HDL-C contributes to vascular homeostasis by preventing monocyte activation. A higher risk of atherosclerotic disease and poor outcomes following cerebrovascular episodes have been associated with lower HDL-C levels.[3,4]

A new biomarker that combines these contrasting biological processes is the monocyte-to-high-density lipoprotein cholesterol ratio (MHR). MHR offers a thorough representation of the inflammatory–atherogenic balance by fusing the pro-inflammatory activities of monocytes with the protective properties of HDL-C. A higher MHR indicates increased inflammatory burden and reduced vascular protection, conditions that may predispose patients to more severe stroke and unfavorable outcomes.[5]

As both monocyte count and HDL-C are routinely measured, inexpensive laboratory parameters, MHR represents a practical and readily available marker for risk stratification in AIS. Emerging evidence suggests that elevated MHR is associated with greater neurological severity, increased risk of complications, prolonged hospitalization, and higher in-hospital mortality. Therefore, MHR may serve as a valuable adjunct to existing clinical and radiological assessments in predicting disease severity and in-hospital outcomes among patients with acute ischemic stroke.[6–8]

AIMS AND OBJECTIVES

The goal of the current study was to assess the Monocyte-to-High-Density Lipoprotein Cholesterol Ratio (MHR) in acute ischaemic stroke patients and ascertain its usefulness as a predictive biomarker. The study specifically sought to correlate MHR values with in-hospital outcomes, such as clinical progression, complications, and death, as well as to evaluate the relationship between MHR and the severity of acute ischaemic stroke upon presentation. By examining this relationship, the study sought to establish whether MHR could serve as a simple, cost-effective, and readily available marker for predicting disease severity and short-term prognosis in patients with acute ischemic stroke.

MATERIALS AND METHODS

Study Design

The study was conducted in the Department of General Medicine at K.R. Hospital, a tertiary care teaching hospital affiliated with Mysore Medical College and Research Institute (MMCRI), Mysuru, Karnataka. The hospital caters to a large number of acute stroke patients and is equipped with advanced diagnostic facilities, including neuroimaging services (NCCT/MRI Brain) and well-established laboratory support. Its high patient inflow and comprehensive stroke care services made it an appropriate setting for the recruitment and evaluation of patients with acute ischemic stroke.

Inclusion and Exclusion Criteria

The study included patients aged 18–80 years who were clinically and radiologically diagnosed with acute ischemic stroke using NCCT Brain or MRI Brain and were admitted within the appropriate time frame from symptom onset. Patients with hemorrhagic stroke or transient ischemic attack (TIA), recent major brain injury, neurosurgery, central nervous system infections, or known malignant tumors were excluded. Additional exclusions comprised patients with severe uncontrolled systemic diseases, dementia, significant psychiatric illness, pre-existing renal or hepatic dysfunction, congestive heart failure (NYHA Class III/IV), chronic inflammatory disorders, chronic infections such as tuberculosis or HIV, known immunological disorders, and those receiving long-term medications that could significantly alter inflammatory markers or lipid profiles, including systemic corticosteroids and antineoplastic agents.

Sample Size Calculation

The sample size was calculated using the formula for estimating a single proportion. The calculated sample size was rounded off to 63 for the purpose of the study.

Z^2PQ/D^2

Where:
S = Required sample size
Z = Standard normal variate at 5% type 1 error (1.96).
_ = Anticipated proportion of the characteristic of interest (prevalence of a relevant stroke outcome marker was estimated at 4% or 0.04 based on prior literature).
Q = 1 – P (0.96)

D = Absolute precision or margin of error (5% or 0.05). The calculation yielded: S = (1.96 *0.04 * 0.96)/(0.05^2) = 59. The sample size was rounded up to 60 participants to account for potential attrition or incomplete date. This was deemed adequate to provide statistically meaningful results for the primary objective of correlation.

Data Collection Procedure

A pre-designed semi-structured proforma was used to gather pertinent demographic, clinical, laboratory, and radiological data following permission from the Institutional Ethics Committee and written informed consent from the patients or their legally designated representatives. The National Institutes of Health Stroke Scale (NIHSS) was used to measure the stroke severity upon admission after a thorough clinical history and clinical examination. Hospital records were used to collect data from routine laboratory testing, such as random blood sugar, lipid profiles, liver and renal function tests, serum electrolytes, and complete blood counts (CBCs). The Monocyte-to-HDL Cholesterol Ratio (MHR) was computed using the absolute monocyte count and HDL cholesterol levels. The diagnosis of acute ischemic stroke was confirmed by neuroimaging (NCCT Brain or MRI Brain). Patients were monitored during their hospital stay, and the Modified Rankin Scale (mRS) and the occurrence of any significant adverse events were used to evaluate in-hospital outcomes upon discharge. All collected data were assigned unique identification numbers to maintain confidentiality and entered into a computerized database for statistical analysis.

Statistical Analysis

Microsoft Excel was used to enter the data, and SPSS software was used for analysis. The data were summarised using descriptive statistics, where continuous variables were expressed as mean ± standard deviation or median (interquartile range) based on data distribution, and categorical variables were expressed as frequencies and percentages. Pearson's or Spearman's correlation analysis was used to evaluate the relationship between the Monocyte-to-HDL Cholesterol Ratio (MHR) and stroke severity (NIHSS score). The Chi-square test or Fisher's exact test were used to assess connections between categorical variables, and the independent samples t-test or Mann-Whitney U test were used to compare outcome groups. The predictive value and ideal cut-off of MHR for severe stroke and poor in-hospital outcomes were found using Receiver Operating Characteristic (ROC) curve analysis. Statistical significance was defined as a p-value of less than 0.05.

RESULTS AND OBSERVATIONS

Variable	Category	Frequency	Percentage (%)
Age	40–50 years	22	34.92
	51–60 years	14	22.22
	61–70 years	13	20.63
	>70 years	14	22.22
Sex	Male	34	53.97
	Female	29	46.03

Table 1. Demographic Characteristics of Study Participants (n=63)

Table 1 illustrates the demographic profile of the study population. Most patients belonged to the 40–50 years age group (34.92%), and there was a slight male predominance (53.97%), indicating that acute ischemic stroke was more common among middle-aged and elderly males.

Risk Factor	Present n (%)	Absent n (%)
Hypertension	29 (46.03)	34 (53.97)
Diabetes Mellitus	39 (61.90)	24 (38.10)
Smoking	19 (30.16)	44 (69.84)
Alcohol Consumption	18 (28.57)	45 (71.43)

Table 2. Distribution of Major Vascular Risk Factors

Table 2 shows the distribution of major vascular risk factors among study participants. Diabetes mellitus was the most common comorbidity (61.9%), followed by hypertension (46.03%), emphasizing the important contribution of metabolic and vascular risk factors in acute ischemic stroke.

CT/MRI Finding	Frequency (n)	Percentage (%)
Anterior Circulation Infarct	18	28.57
PCA Territory Infarct	16	25.40
Lacunar Infarct	15	23.81
MCA Territory Infarct	14	22.22
Total	63	100

Table 3. Neuroimaging Findings in Acute Ischemic Stroke

Table 3 demonstrates the neuroimaging distribution among patients. Anterior circulation infarcts were the most common imaging finding (28.57%), followed by PCA territory infarcts (25.40%).

Variable	Category	Frequency (n)	Percentage (%)
Complications	Nil	28	44.44
	Aspiration Pneumonia	20	31.75
	Sepsis	15	23.81
Clinical Outcome	Improved	25	39.68
	Residual Deficit	20	31.75
	Mortality	18	28.57

Table 4. In-Hospital Complications and Clinical Outcomes

Table 4 summarizes the in-hospital complications and outcomes. Nearly half of the patients had no complications, while aspiration pneumonia was the most common complication. Improvement was observed in

39.68% of patients, whereas mortality occurred in 28.57%.

Complication	N	Mean MHR	SD	Range	p-value
Nil	28	0.01	0.00	0.01–0.02	
Aspiration Pneumonia	20	0.02	0.01	0.01–0.03	
Sepsis	15	0.02	0.00	0.02–0.03	<0.0001

Table 5. Association Between MHR and In-Hospital Complications

Table 5 shows a statistically significant association between MHR and in-hospital complications. Patients who developed aspiration pneumonia or sepsis had higher MHR values compared to those without complications ($p<0.0001$).

Outcome	N	Mean MHR	SD	Range	p-value
Improved	25	0.01	0.00	0.01–0.02	
Residual Deficit	20	0.02	0.01	0.01–0.03	
Mortality	18	0.02	0.00	0.02–0.03	<0.0001

Table 6. Association Between MHR and Clinical Outcome

Table 6 demonstrates that patients with mortality and residual neurological deficits had significantly higher MHR values than patients who improved clinically, indicating the prognostic value of MHR in predicting adverse outcomes.

Variable	Pearson Correlation Coefficient (r)	p-value
MHR vs Hospital Stay Duration	0.82024	<0.0001
MHR vs NIHSS Day 7	0.71709	<0.0001

Table 7. Correlation of MHR with Hospital Stay and Neurological Severity

Table 7 reveals a strong positive correlation between MHR and both duration of hospital stay ($r=0.82024$) and NIHSS score on Day 7 ($r=0.71709$). Higher MHR values were associated with prolonged hospitalization and greater neurological impairment.

Variable	Improved (Mean ± SD)	Mortality (Mean ± SD)	Residual Deficit (Mean ± SD)
NIHSS at Admission	6.12 ± 3.27	20.78 ± 3.10	15.60 ± 4.87
NIHSS Day 7	2.24 ± 2.45	42.00 ± 0.00	14.05 ± 5.31

Table 8. Comparison of NIHSS Scores Across Outcome Groups

Table 8 compares neurological severity across outcome groups. Patients who improved had the lowest NIHSS scores at admission and Day 7, whereas patients who died had the highest scores, demonstrating a strong relationship between stroke severity and clinical outcome.

DISCUSSION

The current study assessed the predictive power of the monocyte-to-high-density lipoprotein cholesterol ratio (MHR) for acute ischaemic stroke patients' in-hospital outcomes and stroke severity. The study population showed a mild male predominance (53.97%), with most patients belonging to the 40–70 year age group. Diabetes mellitus (61.9%) and hypertension (46.03%) were the most common vascular risk factors, highlighting the significant burden of metabolic comorbidities among stroke patients. Similar observations were reported by Sharma and Gandhi, who noted a predominance of vascular risk factors among acute ischemic stroke patients.[9] Wang et al. also demonstrated that increasing age and metabolic risk factors contribute significantly to stroke occurrence.[10]

The baseline laboratory profile in the present study demonstrated elevated leukocyte counts (12.23 ± 3.75 $\times 10^3/\mu L$), increased monocyte counts (0.82 ± 0.29 $\times 10^3/\mu L$), and relatively low HDL levels (40.05 ± 6.52 mg/dL), indicating a pro-inflammatory and atherogenic state. These findings are consistent with the observations of Bolayir et al., who reported higher monocyte counts and lower HDL levels among acute ischemic stroke patients, supporting the role of inflammation and lipid dysregulation in stroke pathogenesis.[11] Similarly, Liu et al. demonstrated significantly elevated monocyte-related inflammatory ratios in stroke patients compared with healthy controls.[12]

A major finding of the present study was the significant association between elevated MHR and stroke severity. Patients with poor outcomes exhibited markedly higher neurological impairment, with admission NIHSS scores of 20.78 ± 3.10 in the mortality group and 15.60 ± 4.87 among patients with residual deficits, compared with 6.12 ± 3.27 among those who improved. An MHR cut-off value of ≥0.018 was found to be associated with severe stroke. These findings support the concept that increased inflammatory burden contributes to greater neurological injury. Sharma et al. reported a significant positive correlation between MHR and NIHSS scores, with mean MHR values of 14.12 ± 2.95 among stroke patients and even higher values (18.48 ± 4.02) among those with clinical deterioration.[13] Likewise, Liu et al. reported that MHR was significantly elevated in ischemic stroke patients and demonstrated good diagnostic performance with an AUC of 0.777.[12]

The present study also demonstrated a clear relationship between elevated MHR and unfavorable in-hospital outcomes. Patients with higher MHR values experienced poorer neurological recovery and increased mortality. These findings are in agreement with Bolayir et al., who identified MHR as an independent predictor of 30-day mortality and reported an optimal cut-off value of 17.52 for mortality prediction.[11] Similarly, Xu et al., in a large cohort of 13,865 patients from the Third China National Stroke Registry, found that patients in the highest MHR quartile had significantly increased risks of mortality (HR 1.45) and poor functional outcomes (OR 1.47) following ischemic stroke.[14]

These results have a well-established biological foundation. By releasing pro-inflammatory cytokines and reactive oxygen species, monocytes contribute to endothelial dysfunction, plaque instability, thrombosis, and neuroinflammation.[15-18] HDL, on the other hand, has endothelial-protective, anti-inflammatory, and antioxidant properties.[19-21] Therefore, MHR represents the balance between inflammatory injury and vascular protection. A higher MHR reflects predominance of inflammatory activity, which may promote infarct expansion, blood-brain barrier disruption, and poor neurological recovery.

The results of the present study are further supported by evidence from other inflammatory biomarkers. Giede-Jeppe et al. demonstrated that elevated neutrophil-to-lymphocyte ratio was associated with unfavourable functional outcomes in acute ischemic stroke.[22] Similarly, Sharma and Gandhi reported a significant correlation between platelet-to-lymphocyte ratio and NIHSS scores.[9] Together, these findings reinforce the critical role of systemic inflammation in determining stroke severity and prognosis.

The findings of the present study indicate that MHR is a simple, inexpensive, and readily available biomarker that can be used for early risk stratification in acute ischemic stroke. The significant association of elevated MHR with greater stroke severity and unfavourable in-hospital outcomes suggests that MHR may serve as a valuable prognostic tool in routine clinical practice.

LIMITATIONS

When evaluating the results, it is important to take into account the many limitations of the current study. The outcomes of this single-center study, which was carried out at a tertiary care hospital, may not be entirely applicable to other demographics or healthcare environments. The statistical power to identify weaker relationships may have been constrained by the very small sample size. Moreover, a conclusive causal association between the monocyte-to-high-density lipoprotein cholesterol ratio (MHR) and stroke severity or outcomes cannot be established due to the observational cross-sectional methodology. Serial measurements that could have offered more prognostic information about the course of the disease were not taken during hospitalisation; instead, only baseline MHR values were evaluated. Furthermore, dietary practices, the use of lipid-lowering drugs, and other inflammatory comorbidities were not thoroughly examined as potential confounding factors. Lastly, the evaluation of the association between MHR and long-term functional recovery, recurrence, or survival following acute ischaemic stroke was limited by the lack of long-term follow-up.

CONCLUSION

The current investigation showed that in patients with acute ischaemic stroke, a higher monocyte-to-high-density lipoprotein cholesterol ratio (MHR) is substantially linked to worse in-hospital outcomes and a more severe stroke. MHR is a straightforward, economical, and clinically valuable biomarker for early risk stratification and prognostic evaluation since it can be easily computed from standard laboratory values. Including MHR in the first assessment of stroke patients may assist identifying those who are more likely to experience unfavourable outcomes, allowing for prompt intervention and optimal management. To confirm these results and investigate the possible relevance of MHR in directing treatment decision-making, further extensive multicenter trials with extended follow-up are necessary.

REFERENCES

1. Amarenco P, Labreuche J. Lipid management in the prevention of stroke: review and updated meta-analysis of statins for stroke prevention. Lancet Neurol 2009;8(5):453-63.
2. Buck BH, Liebeskind DS, Saver JL, et al Early neutrophilia is associated with volume of ischemic tissue in acute stroke. Stroke 2008;39(2):355-60.
3. Zhang W, Zhang XA. Prognostic value of serum lipoprotein(a) levels in patients with acute ischemic stroke. Neuroreport 2014;25(4):262-6.
4. Elkind MS, Luna JM, McClure LA, et al; LIMITS Investigators. C-reactive protein as a prognostic marker after lacunar stroke: levels of inflammatory markers in the treatment of stroke study. Stroke 2014;45(3):707-16.
5. Guo Z, Yu S, Xiao L, et al Dynamic change of neutrophil to lymphocyte ratio and hemorrhagic transformation after thrombolysis in stroke. J Neuroinflammation 2016;13(1):199.
6. Iadecola C, Anrather J. The immunology of stroke: from mechanisms to translation. Nat Med 2011;17(7):796-808.
7. Liberale L, Carbone F, Montecucco F, et al. Statins reduce vascular inflammation in atherogenesis: A review of underlying molecular mechanisms. Int J Biochem Cell Biol 2020;122:105735.
8. Ren H, Liu X, Wang L, et al. Lymphocyte-to-Monocyte Ratio: a novel predictor of the prognosis

of acute ischemic stroke. J Stroke Cerebrovasc Dis 2017;26(11):2595-602.
9. Sharma D, Gandhi N. Role of Platelet to Lymphocyte Ratio (PLR) and its Correlation with NIHSS (National Institute of Health Stroke Scale) for Prediction of Severity in Patients of Acute Ischemic Stroke. J Assoc Physicians India 2021;69(1):56-60.
10. Wang HY, Shi WR, Yi X, et al. Assessing the performance of monocyte to high-density lipoprotein ratio for predicting ischemic stroke: insights from a population-based Chinese cohort. Lipids Health Dis 2019;18(1):127.
11. Bolayir A, Gokce SF, Cigdem B, et al. Monocyte/high-density lipoprotein ratio predicts the mortality in ischemic stroke patients. Neurol Neurochir Pol 2018;52(2):150-5.
12. Liu H, Zhan F, Wang Y. Evaluation of monocyte-to-high-density lipoprotein cholesterol ratio and monocyte-to-lymphocyte ratio in ischemic stroke. J Int Med Res 2020;48(7):300060520933806.
13. Sharma D, Aravind S, Joseph S, et al. The role of monocyte to high-density lipoprotein cholesterol ratio in predicting the severity of acute ischemic stroke and its association with the NIHSS. J Assoc Physicians India 2023;71(8):11-2.
14. Xu Q, Wu Q, Chen L, et al Monocyte to high-density lipoprotein ratio predicts clinical outcomes after acute ischemic stroke or transient ischemic attack. CNS Neurosci Ther 2023;29(7):1953-64.
15. Swirski FK, Libby P, Aikawa E, et al monocytes dominate hypercholesterolemia-associated monocytosis and give rise to macrophages in atheromata. J Clin Invest 2007;117(1):195-205.
16. Tacke F, Alvarez D, Kaplan TJ, et al Monocyte subsets differentially employ CCR2, CCR5, and CX3CR1 to accumulate within atherosclerotic plaques. J Clin Invest 2007;117(1):185-94.
17. Soehnlein O, Drechsler M, Döring Y, et al Distinct functions of chemokine receptor axes in the atherogenic mobilization and recruitment of classical monocytes. EMBO Mol Med 2013;5(3):471-81.
18. Gliem M, Mausberg AK, Lee JI, et al Macrophages prevent hemorrhagic infarct transformation in murine stroke models. Ann Neurol 2012;71(6):743-52.
19. Gordon T, Castelli WP, Hjortland MC, et al. High density lipoprotein as a protective factor against coronary heart disease. The Framingham Study. Am J Med 1977;62(5):707-14.
20. Mineo C, Deguchi H, Griffin JH, et al. Endothelial and antithrombotic actions of HDL. Circ Res 2006;98(11):1352-64.
21. Murphy AJ, Woollard KJ, Hoang A, et al High-density lipoprotein reduces the human monocyte inflammatory response. Arterioscler Thromb Vasc Biol 2008;28(11):2071-7.
22. Giede-Jeppe A, Madžar D, Sembill JA, et al Increased neutrophil-to-lymphocyte ratio is associated with unfavorable functional outcome in acute ischemic stroke. Neurocrit Care 2020;33(1):97-104.