

Monocyte-to-High-Density Lipoprotein Cholesterol Ratio and Clinical Angiographic Risk Stratification in Patients with ST Segment Elevation Myocardial Infarction

Authors:

Deepa .K¹, Meghana V², Nikhil .M B^{3*}

¹Assistant Professor, Department of General Medicine, Subbaiah Institute of Medical Sciences And Research Institute Shivamogga, Karnataka, India.

²Senior Resident, Department of General Medicine, Subbaiah Institute of Medical Sciences And Research Institute Shivamogga, Karnataka, India.

³Assistant Professor, Department of General Medicine, Subbaiah Institute of Medical Sciences, Shivamogga, Karnataka, India.

***Corresponding Author:**

Dr. Nikhil .M B, Assistant Professor, Department of General Medicine, Subbaiah Institute of Medical Sciences, Shivamogga, Karnataka, India

DOI: <https://doi.org/10.5281/zenodo.21989824>

Article Received: 23 July 2026, Accepted: 13 August 2026, Online First: 18 August 2026

ABSTRACT

Background: The monocyte-to-high-density lipoprotein cholesterol ratio (MHR) combines a pro-inflammatory cellular marker with an anti-inflammatory lipid marker and may reflect cardiovascular disease burden. **Methods:** This secondary analysis used the existing prospective observational cohort of 103 participants admitted to KIMS Hospital, Hubballi. Twenty-eight participants had normal coronary arteries and 75 had significant coronary angiographic disease. STEMI patients were categorized into low-risk (TIMI 0–2; n=27) and high-risk (TIMI ≥3; n=48) groups. Clinical, laboratory and angiographic variables were evaluated. **Results:** Mean MHR increased progressively from 1.7±0.05 in controls to 2.4±0.11 in low-risk STEMI and 3.3±0.03 in high-risk STEMI (p<0.001). Monocyte count increased while HDL-C decreased across the same groups. SVD predominated in the low-risk group (59.3%), whereas DVD predominated in the high-risk group (66.7%); TVD was present in 18.8% of high-risk patients (p<0.001). **Conclusion:** Higher MHR was associated with increasing clinical risk and a more extensive coronary angiographic disease pattern. MHR may be a useful adjunct to established risk assessment, although independent prognostic value requires prospective validation.

Keywords: STEMI; MHR; TIMI score; PCI; CAD; CAG; inflammation; multivessel coronary disease.

INTRODUCTION

ST-segment elevation myocardial infarction (STEMI) remains an important cause of morbidity and mortality in patients with coronary artery disease. Early diagnosis, reperfusion and risk stratification are essential because patients with STEMI are at increased risk of complications. Rapid diagnosis of acute myocardial infarction is critical because early medical intervention and revascularization provide the greatest benefit within the first few hours. The TIMI risk score is a widely used clinical model for risk stratification in STEMI.

Atherosclerosis is a chronic inflammatory disease characterized by lipid accumulation, endothelial dysfunction and inflammatory cell recruitment.

Monocytes contribute to plaque formation, progression and instability, while HDL-C has anti-inflammatory, antioxidant and cholesterol-efflux properties.

The monocyte-to-HDL cholesterol ratio (MHR) has emerged as an inexpensive marker integrating inflammatory and lipid-related pathways. Previous studies have associated higher MHR with adverse cardiovascular outcomes and coronary disease severity.

The original publication from this cohort evaluated the association of MHR with TIMI risk score. The present manuscript is framed as a secondary analysis focusing on the additional relationship between MHR, clinical severity and coronary angiographic disease burden.

AIM

To evaluate the association of admission MHR with clinical risk category and coronary angiographic severity in patients presenting with acute STEMI.

OBJECTIVES

1. To compare MHR between participants with STEMI with normal coronary arteries and with significant coronary artery disease.
2. To describe the relationship between clinical risk category and angiographic disease pattern.
3. To compare monocyte count and HDL-C across study groups.

MATERIALS AND METHODS

This is a single-centre prospective observational cohort secondary analysis conducted under the Department of General Medicine, KIMS Hubballi.

Patients admitted to ICCU, KIMS Hubballi with clinical and ECG features suggestive of acute STEMI were considered. A total of 103 participants were included in the original cohort. Of these, 75 had STEMI with significant coronary angiographic findings and 28 had normal coronary arteries and served as controls.

RESULTS

Baseline characteristics: There were 78 (75.72%) males and 25 (24.27%) females. The mean age of the cohort was approximately 60.8 years. Diabetes mellitus was reported in 26.21% and hypertension in 45.63% of participants.

Table 1. Sex distribution across study groups

Group	n	Male	Female
Control	28	19 (67.9%)	9 (32.1%)
Low-risk STEMI	27	22 (81.5%)	5 (18.5%)
High-risk STEMI	48	37 (77.1%)	11 (22.9%)

Table 2. Comparison of TIMI score across groups

Group	Total	Mean	SD	P value
Control	28	0	0	<0.001
Low risk	27	2	0	
High risk	48	4	0.7	

The mean (SD) TIMI score in the control, low-risk and high-risk groups was 0 (0), 2 (0) and 4 (0.7), respectively. There was a significant difference in mean TIMI scores across the three study groups ($p < 0.001$).

Table 3. Distribution of Killip class

Killip class	Control group N (%)	Low-risk group N (%)	High-risk group N (%)	P value
I	24 (85.7)	22 (81.5)	40 (83.3)	0.71
II	2 (7.1)	4 (14.8)	4 (8.3)	
III	2 (7.1)	1 (3.7)	2 (4.2)	
IV	0 (0)	0 (0)	2 (4.2)	
Total	28 (100)	27 (100)	48 (100)	

More than 80% of participants had Killip class I in all three groups (85.7%, 81.5% and 83.3%). Killip class II was the second most common class. No participant in the control or low-risk group had class IV, whereas two patients (4.2%) in the high-risk group had class IV; the difference was not statistically significant ($p = 0.71$).

Inclusion Criteria: Age >18 years; informed consent; clinical and ECG features of acute STEMI; and coronary angiography.

Exclusion Criteria: Previous coronary revascularization; active infection; severe liver disease; hematological disorder; inflammatory or infectious disease.

Investigations performed included complete hemogram, renal and liver function tests, lipid profile, ECG, 2D echocardiography and coronary angiography.

STEMI patients were divided into low-risk (TIMI 0–2) and high-risk (TIMI ≥ 3) groups. MHR was calculated from the admission monocyte count and HDL-C concentration.

Data were analyzed using SPSS version 17.0. Qualitative variables were expressed as frequencies and percentages. Quantitative variables were expressed as mean $\pm$ SD or median/range depending on distribution. Chi-square testing was used for categorical variables and ANOVA for continuous laboratory variables. A p value <0.05 was considered statistically significant.

Table 4. Coronary angiographic pattern among STEMI risk groups

Angiographic pattern	Low risk, n (%)	High risk, n (%)
SVD	16 (59.3)	7 (14.6)
DVD	9 (33.3)	32 (66.7)
TVD	2 (7.4)	9 (18.8)

Overall angiographic distribution differed significantly ($p<0.001$). SVD predominated in the low-risk group, while DVD and TVD were more frequent in the high-risk group.

Table 5. Lipid and inflammatory parameters across study groups

Parameter	Control	Low risk	High risk	P value
Total cholesterol (mg/dL)	191.5 $\pm$ 4.3	216.6 $\pm$ 10.1	219.2 $\pm$ 7.4	<0.001
HDL-C (mg/dL)	44.0 $\pm$ 1.0	42.9 $\pm$ 1.2	40.9 $\pm$ 0.9	<0.001
Monocyte count ($\times 10^3/\mu\text{L}$)	0.76 $\pm$ 0.02	0.82 $\pm$ 0.02	1.00 $\pm$ 0.01	<0.001
MHR	1.7 $\pm$ 0.05	2.4 $\pm$ 0.11	3.3 $\pm$ 0.03	<0.001

DISCUSSION

The principal finding of this secondary analysis is the stepwise increase in MHR from participants with normal coronary arteries to low-risk and high-risk STEMI. This pattern was accompanied by increasing monocyte counts and decreasing HDL-C. Monocytes are important mediators of vascular inflammation and plaque progression, whereas HDL-C contributes to cholesterol efflux and has anti-inflammatory effects. The combined ratio therefore provides a biologically plausible measure of the balance between inflammatory activity and protective lipid effects. The angiographic findings provide an additional clinically relevant observation. SVD predominated in low-risk STEMI, whereas DVD and TVD were substantially more frequent in the high-risk group. The clinical risk gradient was therefore accompanied by a greater coronary disease burden. Previous studies in the thesis literature have reported associations between MHR and cardiovascular outcomes, coronary disease severity, stent thrombosis and mortality. The present analysis is concordant with these observations but extends the focus of the original cohort toward angiographic disease distribution. MHR has practical advantages because both components are routinely available at admission. Nevertheless, the present study cannot establish MHR as an independent predictor because adjusted patient-level regression and ROC analysis were not performed. These analyses should be undertaken before claiming independent diagnostic or prognostic utility.

Clinical Implications

MHR may be used as a simple adjunctive marker during initial assessment of STEMI. A higher value may identify patients with greater inflammatory burden and more extensive coronary disease. It should complement, rather than replace, established risk scores and angiographic assessment.

Strengths of the Study

Strengths: The cohort includes clinical, laboratory and coronary angiographic information with comparison across normal coronary, low-risk STEMI and high-risk STEMI groups.

Limitation

Small sample size, single-centre design, single admission MHR measurement, limited longitudinal outcome data and secondary analysis of a previously published cohort.

Future Research and Recommendations

Larger multicentre prospective studies with longer follow-up and standardized MHR assessment are recommended to validate its independent prognostic value and determine its usefulness in predicting coronary angiographic severity and cardiovascular outcomes in STEMI patients.

CONCLUSION

Higher MHR was associated with increasing clinical risk and a more extensive coronary angiographic disease pattern in patients with STEMI. High-risk patients had higher monocyte counts, lower HDL-C and a greater prevalence of multivessel disease. MHR is a promising, inexpensive adjunct marker, but larger prospective studies with multivariable and ROC analyses are required to determine its independent clinical utility.

DECLARATIONS

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Institutional Ethics Committee of KIMS Hospital, Hubballi, prior to commencement of the study.

Written informed consent was obtained from all participants.

Consent for Publication

Not applicable, as no identifiable individual patient information or images are presented.

Availability of Data and Materials

The data generated and/or analysed during the study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

Funding

No funding was received for this study.

Authors' Contributions

All authors contributed to the study conception and design, data collection, analysis and interpretation, manuscript preparation, and critical revision. All authors approved the final version of the manuscript.

REFERENCES

1. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR. ESC/ACCF/AHA/WHF Expert Consensus Document. Circulation. 2012;126(16):2020-35.
2. Lee KL, Woodlief LH, Topol EJ, et al. Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction. Results from an international trial of 41,021 patients. GUSTO-I Investigators. Circulation. 1995;91:1659-68.
3. Amin ST, Morrow DA, Braunwald E, Sloan S, Contant C, Murphy S, Antman EM. Dynamic TIMI risk score for STEMI. Journal of the American Heart Association. 2013 Jan 29;2(1):e003269.
4. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002 Mar 5;105(9):1135-43.
5. Ross R, Harker L. Hyperlipidemia and atherosclerosis. Science. 1976 Sep 17;193(4258):1094-100.
6. Spady DK. Reverse cholesterol transport and atherosclerosis regression. Circulation. 1999 Aug 10;100(6):576-8.
7. Favari E, Chroni A, Tietge UJ, Zanotti I, Bernini F. Cholesterol efflux and reverse cholesterol transport. High density lipoproteins. 2015:181-206.
8. Barter P. The role of HDL-cholesterol in preventing atherosclerotic disease. European heart journal Supplements. 2005 Jul 1;7(suppl_F):F4-8.
9. Ancuta P, Wang J, Gabuzda D. CD16+ monocytes produce IL-6, CCL2, and matrix metalloproteinase-9 upon interaction with CX3CL1-expressing endothelial cells. Journal of leukocyte biology. 2006 Nov;80(5):1156-64.
10. Canpolat U, Aytemir K, Yorgun H, Şahiner L, Kaya EB, Çay S, Topaloğlu S, Aras D, Oto A. The role of preprocedural monocyte-to-high-density lipoprotein ratio in prediction of atrial fibrillation recurrence after cryoballoon-based catheter ablation. Ep Europace. 2015 Dec 1;17(12):1807-15.
11. Cetin EH, Cetin MS, Canpolat U, Aydin S, Topaloglu S, Aras D, Aydogdu S. Monocyte/HDL-cholesterol ratio predicts the definite stent thrombosis after primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. Biomarkers in medicine. 2015 Oct;9(10):967-77.
12. Karataş MB, Çanga Y, Özcan KS, İpek G, Güngör B, Onuk T, Durmuş G, Öz A, Karaca M, Bolca O. Monocyte to high-density lipoprotein ratio as a new prognostic marker in patients with STEMI undergoing primary percutaneous coronary intervention. The American journal of emergency medicine. 2016 Feb 1;34(2):240-4.
13. Olivares R, Ducimetiere P, Claude JR. Monocyte count: a risk factor for coronary heart disease?. American journal of epidemiology. 1993 Jan 1;137(1):49-53.
14. Zhang Y, Li S, Guo Y-L, Wu N-Q, Zhu C-G, Gao Y, et al. Is monocyte to HDL ratio superior to monocyte count in predicting the cardiovascular outcomes: evidence from a large cohort of Chinese patients undergoing coronary angiography. Ann Med. 2016;48(5):305-12.
15. Canpolat U, Çetin EH, Cetin S, Aydin S, Akboga MK, Yayla C, et al. Association of Monocyte-to-HDL Cholesterol Ratio with Slow Coronary Flow is Linked to Systemic Inflammation. Clin Appl Thromb. 2016;22(5):476-82.
16. Cetin MS, Ozcan Cetin EH, Kalender E, Aydin S, Topaloglu S, Kisacik HL, 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):1077-86.