ASSOCIATION OF INFLAMMATORY MARKERS WITH DISEASE SEVERITY AND IN-HOSPITAL OUTCOMES IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION: A HOSPITAL-BASED OBSERVATIONAL STUDY FROM BIHAR

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ABSTRACT

Background: Acute myocardial infarction (AMI) remains a major cause of morbidity and mortality worldwide. Increasing evidence suggests that inflammation plays a crucial role in the initiation, progression, and clinical manifestations of acute coronary syndromes. Inflammatory biomarkers may provide valuable prognostic information in patients with AMI and aid in early risk stratification.

Objectives: To assess the levels of inflammatory markers in patients with acute myocardial infarction and determine their association with disease severity and in-hospital outcomes.

Methods: A hospital-based observational analytical cross-sectional study was conducted in a tertiary care hospital in Bihar from February 2022 to July 2022. A total of 66 adult patients diagnosed with acute myocardial infarction were enrolled using consecutive sampling. Clinical and demographic data were recorded using a structured case record form. Inflammatory markers including C-reactive protein (CRP), total leukocyte count (TLC), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR) were assessed within 24 hours of admission. Left ventricular ejection fraction and in-hospital outcomes were evaluated. Statistical analysis was performed using SPSS version 25.0. A p-value <0.05 was considered statistically significant.

Results: Among the 66 patients, 66.7% were males and 68.2% had ST-elevation myocardial infarction. Hypertension was the most common cardiovascular risk factor. Mean CRP, TLC, ESR, and NLR values were elevated among study participants. Patients with left ventricular ejection fraction below 45% demonstrated significantly higher CRP, TLC, ESR, and NLR levels compared with those having preserved ventricular function. Similarly, patients who developed in-hospital complications had significantly elevated inflammatory marker levels. CRP and NLR showed the strongest associations with ventricular dysfunction and adverse clinical outcomes. Heart failure was

the most frequent complication, followed by arrhythmias and cardiogenic shock. In-hospital mortality was observed in a small proportion of patients.

Conclusion: Inflammatory markers are significantly associated with disease severity and adverse in-hospital outcomes in patients with acute myocardial infarction. CRP and NLR, in particular, may serve as useful, cost-effective adjunctive biomarkers for early risk assessment and prognostication. Incorporation of these markers into routine clinical evaluation may improve identification of high-risk patients and facilitate timely management.

Keywords: Acute myocardial infarction; C-reactive protein; Inflammation; Neutrophil-to-lymphocyte ratio; Prognosis; Cardiovascular disease; Risk stratification.

INTRODUCTION

Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality worldwide despite substantial advances in preventive cardiology, diagnostic modalities, and therapeutic interventions. Cardiovascular diseases account for a significant proportion of global deaths, with ischemic heart disease being the predominant contributor. The burden of AMI continues to increase in low- and middle-income countries owing to urbanization, changing lifestyles, increasing prevalence of diabetes mellitus, hypertension, obesity, and tobacco consumption [1]. Early identification of patients at higher risk of adverse outcomes is essential for improving clinical management and reducing mortality.

The pathogenesis of AMI is complex and involves not only atherosclerotic plaque formation but also inflammatory processes that contribute to plaque instability, rupture, thrombosis, and myocardial injury [2]. Inflammation plays a pivotal role throughout the spectrum of coronary artery disease, from endothelial dysfunction and atherogenesis to the occurrence of acute coronary events [3]. Following plaque rupture, activation of inflammatory pathways leads to recruitment of leukocytes and release of cytokines, which further amplify myocardial damage and influence the healing process [4].

Several inflammatory biomarkers have been investigated as potential indicators of disease severity and prognosis in patients with AMI. Among these, C-reactive protein (CRP), total leukocyte count (TLC), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR) have received considerable attention due to their accessibility and cost-effectiveness [5]. Elevated levels of these markers have been associated with larger infarct size, impaired ventricular function, recurrent ischemic events, and increased mortality in various clinical studies [6]. Assessment of inflammatory markers may therefore provide additional information beyond conventional clinical and biochemical parameters.

South Asian populations, particularly those in India, experience cardiovascular disease at a younger age and often exhibit more severe clinical manifestations compared to Western populations [7]. India has witnessed a rapid rise in the prevalence of coronary artery disease over recent decades, creating a substantial public health burden. The increasing incidence of AMI among economically productive age groups further emphasizes the need for improved risk stratification and early prognostic assessment [8]. In resource-limited healthcare settings, inexpensive laboratory markers that can aid clinical decision-making are of particular value.

Although several international studies have demonstrated associations between inflammatory markers and AMI outcomes, regional variations in genetic, environmental, socioeconomic, and healthcare-related factors may influence these relationships [9]. Furthermore, data from tertiary care

hospitals in Bihar remain relatively limited, and there is a need for local evidence regarding the clinical utility of routinely measured inflammatory biomarkers in patients presenting with AMI. Understanding these associations may facilitate early identification of high-risk patients and contribute to more individualized management strategies.

Given the growing recognition of inflammation as a central mechanism in acute coronary syndromes and the limited regional data available, the present study was undertaken to evaluate the role of inflammatory markers in patients with acute myocardial infarction admitted to a tertiary care hospital in Bihar. The objectives of the study were to assess the levels of selected inflammatory markers among patients with AMI and to determine their association with clinical severity and in-hospital outcomes.

METHODOLOGY

Study Design: A hospital-based observational analytical cross-sectional study was conducted to evaluate the role of inflammatory markers in patients diagnosed with acute myocardial infarction (AMI).

Study Setting: The study was conducted in the Department of General Medicine and Coronary Care Unit (CCU) of a tertiary care teaching hospital in Bihar. The hospital serves as a referral center for urban and rural populations and manages a substantial number of cardiovascular emergencies.

Study Duration: The study was conducted over a period of six months from February 2022 to July 2022.

Study Population: The study population comprised adult patients admitted with a diagnosis of acute myocardial infarction during the study period. Diagnosis of AMI was established based on clinical presentation, electrocardiographic findings, and elevated cardiac biomarkers in accordance with contemporary diagnostic criteria.

Inclusion Criteria

Patients aged ≥18 years.
Patients diagnosed with acute myocardial infarction (ST-elevation myocardial infarction [STEMI] or non-ST-elevation myocardial infarction [NSTEMI]).
Patients admitted within 24 hours of symptom onset.
Patients willing to provide written informed consent.

Exclusion Criteria

Patients with known chronic inflammatory or autoimmune disorders.
Patients with acute or chronic infectious diseases at presentation.
Patients with malignancy.
Patients receiving corticosteroids, immunosuppressive agents, or anti-inflammatory therapy for chronic conditions.
Patients with severe hepatic disease or end-stage renal disease.
Patients unwilling to participate in the study.

Sample Size: The sample size was calculated using the formula for estimation of a proportion:

$$n = \frac{Z^2 pq}{d^2}$$

Where:

(n) = required sample size
(Z) = 1.96 at 95% confidence level
(p) = anticipated prevalence of elevated inflammatory markers among AMI patients (assumed as 50% due to variable estimates in literature)
(q = 1-p = 50%)
(d) = absolute precision of 12%

$$n = \frac{(1.96)^2 \times 0.5 \times 0.5}{(0.12)^2}$$

n = 66

The calculated sample size was approximately 66 participants. Therefore, a total of 66 eligible patients were enrolled in the study.

Sampling Technique: Consecutive sampling was employed. All eligible patients admitted during the study period who fulfilled the inclusion criteria and provided informed consent were recruited until the required sample size of 66 was achieved.

Data Collection Tools and Procedure: After obtaining informed consent, detailed demographic and clinical information was collected using a predesigned and pretested case record form. Data included age, sex, cardiovascular risk factors, presenting symptoms, type of myocardial infarction, and relevant medical history. Venous blood samples were obtained within 24 hours of admission for assessment of inflammatory markers including C-reactive protein (CRP), total leukocyte count (TLC), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR). Routine investigations such as cardiac biomarkers, electrocardiography, and echocardiography were performed as part of standard patient care. Patients were monitored during hospitalization, and in-hospital complications including heart failure, arrhythmias, cardiogenic shock, and mortality were documented.

Study Variables: The independent variables included demographic characteristics (age, sex), cardiovascular risk factors (hypertension, diabetes mellitus, smoking status, dyslipidemia), type of myocardial infarction, and inflammatory markers (CRP, TLC, ESR, and NLR). The dependent variables included indicators of disease severity and in-hospital outcomes such as left ventricular ejection fraction, occurrence of heart failure, arrhythmias, cardiogenic shock, duration of hospitalization, and in-hospital mortality. The study primarily assessed the association between inflammatory marker levels and clinical outcomes among patients with AMI.

Statistical Analysis: Data were entered into Microsoft Excel and analyzed using the Statistical Package for the Social Sciences (SPSS) software version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ±standard deviation or median with interquartile range, depending on data distribution. Categorical variables were presented as frequencies and percentages. Student's t-test or Mann–Whitney U test was used for comparison of continuous variables, while Chi-square test or Fisher's exact test was applied for categorical variables. Correlation analysis was performed to assess relationships between inflammatory markers and measures of disease severity. A p-value <0.05 was considered statistically significant.

Ethical Considerations: Written informed consent was obtained from all participants before enrollment. Confidentiality and anonymity of patient information were maintained throughout the

study. Participation was voluntary, and patients were free to withdraw from the study at any stage without affecting their treatment. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki and its subsequent amendments.

RESULTS

Sixty-six patients with acute myocardial infarction were enrolled in the study. The majority were males and belonged to the 50–69-year age group. Hypertension was the most prevalent cardiovascular risk factor, followed by smoking/tobacco use, dyslipidemia, and diabetes mellitus. STEMI constituted more than two-thirds of the cases (Table 1).

Table 1. Baseline demographic and clinical characteristics of study participants (n=66)

Variable	Category	Frequency (n)	Percentage (%)
Age (years)	<50	15	22.7
	50–59	21	31.8
	60–69	19	28.8
	≥70	11	16.7
Sex	Male	44	66.7
	Female	22	33.3
Hypertension	Present	36	54.5
	Absent	30	45.5
Diabetes mellitus	Present	24	36.4
	Absent	42	63.6
Smoking/Tobacco use	Present	29	43.9
	Absent	37	56.1
Dyslipidemia	Present	26	39.4
	Absent	40	60.6
Type of AMI	STEMI	45	68.2
	NSTEMI	21	31.8

Inflammatory markers including CRP, TLC, ESR, and NLR were elevated among study participants, while the mean left ventricular ejection fraction indicated mild impairment of systolic function (Table 2).

Table 2. Inflammatory marker profile among patients with acute myocardial infarction (n=66)

Parameter	Unit	Mean ± SD
C-reactive protein (CRP)	mg/L	19.4 ± 8.7
Total leukocyte count (TLC)	$\times 10^3$ cells/μL	11.8 ± 3.1
Erythrocyte sedimentation rate (ESR)	mm/hour	28.6 ± 10.5
Neutrophil-to-lymphocyte ratio (NLR)	Ratio	4.9 ± 1.8
Left ventricular ejection fraction (LVEF)	%	47.8 ± 8.9

Patients with left ventricular systolic dysfunction demonstrated significantly higher levels of all evaluated inflammatory markers compared with those having preserved ventricular function. The strongest associations were observed for CRP and NLR (Table 3).

Table 3. Association of inflammatory markers with left ventricular systolic dysfunction

Variable	LVEF <45% (n=26) Mean ± SD	LVEF ≥45% (n=40) Mean ± SD	p-value
CRP (mg/L)	24.2 ± 8.3	16.3 ± 6.7	<0.001
TLC (×10³ cells/μL)	13.1 ± 3.0	10.9 ± 2.7	0.006
ESR (mm/hour)	32.8 ± 10.9	25.9 ± 8.8	0.009
NLR	6.0 ± 1.9	4.2 ± 1.3	<0.001

Similarly, patients who developed in-hospital complications had significantly elevated inflammatory marker levels compared with those without complications. Increased CRP and NLR showed particularly strong associations with adverse clinical outcomes (Table 4).

Table 4. Association between inflammatory markers and in-hospital complications

Variable	Complications Present (n=20) Mean ± SD	Complications Absent (n=46) Mean ± SD	p-value
CRP (mg/L)	25.1 ± 8.6	16.9 ± 6.8	<0.001
TLC (×10³ cells/μL)	13.4 ± 3.2	11.1 ± 2.8	0.004
ESR (mm/hour)	33.7 ± 10.4	26.4 ± 9.1	0.007
NLR	6.1 ± 1.7	4.4 ± 1.4	<0.001

Complications included heart failure, clinically significant arrhythmias, and cardiogenic shock.

Heart failure was the most common in-hospital complication, followed by arrhythmias and cardiogenic shock. Overall in-hospital mortality was low, and the majority of patients experienced no major complications during hospitalization (Table 5).

Table 5. Distribution of in-hospital outcomes among patients with acute myocardial infarction (n=66)

Outcome	Frequency (n)	Percentage (%)
Heart failure	12	18.2
Arrhythmias	8	12.1
Cardiogenic shock	4	6.1
In-hospital mortality	3	4.5
No major complication	39	59.1

DISCUSSION

The present hospital-based observational study evaluated the role of inflammatory markers in patients with acute myocardial infarction admitted to a tertiary care hospital in Bihar. The principal findings of the study were that inflammatory markers, namely C-reactive protein (CRP), total leukocyte count (TLC), erythrocyte sedimentation rate (ESR), and neutrophil-to-lymphocyte ratio (NLR), were elevated among patients with AMI. Furthermore, higher levels of these markers were significantly associated with left ventricular systolic dysfunction and adverse in-hospital outcomes, including heart

failure, arrhythmias, and cardiogenic shock. Among the evaluated markers, CRP and NLR demonstrated the strongest associations with disease severity and complications.

The demographic profile observed in the present study is consistent with the established epidemiology of AMI in South Asian populations. Most patients were males and belonged to the middle-aged and elderly age groups. Similar demographic patterns have been reported in previous studies from India and other developing countries, where cardiovascular risk factors such as hypertension, diabetes mellitus, smoking, and dyslipidemia contribute substantially to the burden of coronary artery disease [7,8]. The predominance of STEMI observed in the present study also aligns with findings from several tertiary care centers in India, where delayed presentation and limited access to preventive healthcare services continue to influence disease patterns.

Inflammation is increasingly recognized as a fundamental component of atherosclerosis and acute coronary syndromes. Plaque rupture initiates a cascade of inflammatory responses involving activation of leukocytes, release of cytokines, endothelial dysfunction, and thrombus formation [2,3]. Consequently, inflammatory biomarkers may reflect the intensity of myocardial injury and the extent of ongoing vascular inflammation. The elevated inflammatory marker levels observed in the present study support this biological relationship and are consistent with current understanding of AMI pathophysiology.

The significant association between elevated CRP levels and reduced left ventricular ejection fraction observed in this study is in agreement with previous investigations. CRP is an acute-phase reactant synthesized by the liver in response to inflammatory cytokines, particularly interleukin-6. Higher CRP concentrations have been associated with larger infarct size, impaired ventricular remodeling, and increased risk of subsequent cardiovascular events [6,10]. The findings of the present study further support the utility of CRP as a readily available marker for identifying patients at increased risk of adverse outcomes following AMI.

Similarly, elevated TLC was associated with left ventricular dysfunction and in-hospital complications. Leukocytosis is considered a marker of systemic inflammatory activation and has been linked to endothelial injury, microvascular obstruction, and infarct expansion [11]. Previous studies have demonstrated that increased leukocyte counts are independently associated with mortality and major adverse cardiovascular events after AMI. The present findings reinforce the prognostic relevance of this inexpensive and routinely available laboratory parameter.

The neutrophil-to-lymphocyte ratio emerged as one of the strongest predictors of adverse clinical outcomes in the current study. NLR integrates information regarding both innate and adaptive immune responses and has gained considerable attention as a marker of systemic inflammation. Elevated NLR has been associated with increased infarct size, reduced myocardial perfusion, heart failure, and mortality among patients with acute coronary syndromes [12,13]. The strong association observed between NLR and adverse outcomes in this study is therefore biologically plausible and consistent with existing evidence.

ESR was also significantly elevated among patients with greater disease severity and complications. Although ESR is a nonspecific marker of inflammation, previous investigations have suggested that persistently elevated ESR may indicate ongoing inflammatory activity and unfavorable cardiovascular prognosis [14]. While ESR lacks the specificity of CRP or NLR, its widespread availability and low cost may make it useful as an adjunctive marker in resource-constrained settings.

From a clinical perspective, the findings of this study suggest that inflammatory markers may complement conventional diagnostic and prognostic tools in patients with AMI. Early identification of patients at higher risk for ventricular dysfunction and complications could facilitate closer monitoring, timely intervention, and optimization of treatment strategies. In low- and middle-income countries such as India, where access to advanced prognostic biomarkers may be limited, inexpensive inflammatory markers could provide valuable additional information for risk stratification.

The strengths of the present study include the prospective collection of clinical and laboratory data, inclusion of routinely available inflammatory markers, and evaluation of their relationship with clinically relevant outcomes. The study also provides region-specific evidence from a tertiary care hospital in Bihar, contributing to the limited local literature on this topic.

However, several limitations should be acknowledged. The study was conducted at a single center and included a relatively modest sample size, which may limit the generalizability of the findings. The observational design precludes establishment of causal relationships between inflammatory markers and outcomes. Inflammatory markers were assessed during hospitalization, and serial measurements were not performed. Long-term follow-up was also beyond the scope of the study, preventing assessment of their association with post-discharge cardiovascular events.

Overall, the findings support the growing body of evidence that systemic inflammation plays an important role in the clinical course of acute myocardial infarction. Routine inflammatory markers, particularly CRP and NLR, may serve as useful adjuncts for early risk assessment and prognostication in patients presenting with AMI.

CONCLUSION

The present study demonstrated that inflammatory markers, including C-reactive protein, total leukocyte count, erythrocyte sedimentation rate, and neutrophil-to-lymphocyte ratio, were elevated among patients with acute myocardial infarction and showed significant associations with disease severity and adverse in-hospital outcomes. Patients with left ventricular systolic dysfunction and those who developed complications during hospitalization exhibited higher inflammatory marker levels compared with patients who had a more favorable clinical course.Among the evaluated biomarkers, C-reactive protein and neutrophil-to-lymphocyte ratio demonstrated particularly strong associations with ventricular dysfunction and in-hospital complications, suggesting their potential utility in early risk stratification. These markers are inexpensive, readily available, and can be incorporated into routine clinical assessment, especially in resource-constrained healthcare settings.
The findings support the role of inflammation in the pathophysiology and progression of acute myocardial infarction and highlight the potential value of inflammatory markers as adjunctive prognostic tools. Larger multicentric studies with long-term follow-up are recommended to further validate these observations and establish their role in guiding clinical decision-making and patient management.

DECLARATIONS

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Conflict of Interest: The authors declare that there is no conflict of interest.
Informed Consent: Written informed consent was obtained from all participants prior to enrollment in the study.

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REFERENCES

1. Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, Himmelfarb CD, Khera A, Lloyd-Jones D, McEvoy JW, Michos ED. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2019 Sep 10;74(10):1376-414.
2. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002 Mar 5;105(9):1135-43.
3. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. New England journal of medicine. 2005 Apr 21;352(16):1685-95.
4. Frangogiannis NG. The inflammatory response in myocardial injury, repair, and remodelling. Nature Reviews Cardiology. 2014 May;11(5):255-65.
5. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon III RO, Criqui M, Fadl YY, Fortmann SP, Hong Y, Myers GL, Rifai N. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. circulation. 2003 Jan 28;107(3):499-511.
6. Ridker PM. Clinical application of C-reactive protein for cardiovascular disease detection and prevention. Circulation. 2003 Jan 28;107(3):363-9.
7. Joshi P, Islam S, Pais P, Reddy S, Dorairaj P, Kazmi K, Pandey MR, Haque S, Mendis S, Rangarajan S, Yusuf S. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. Jama. 2007 Jan 17;297(3):286-94.
8. Gupta R, Mohan I, Narula J. Trends in coronary heart disease epidemiology in India. Annals of global health. 2016 Mar 1;82(2):307-15.
9. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, White HD, Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction. Fourth universal definition of myocardial infarction (2018). Journal of the American college of cardiology. 2018 Oct 30;72(18):2231-64.
10. Suleiman M, Khatib R, Agmon Y, Mahamid R, Boulos M, Kapeliovich M, Levy Y, Beyar R, Markiewicz W, Hammerman H, Aronson D. Early inflammation and risk of long-term development of heart failure and mortality in survivors of acute myocardial infarction: predictive role of C-reactive protein. Journal of the American College of Cardiology. 2006 Mar 7;47(5):962-8.
11. Núñez J, Núñez E, Bodí V, Sanchis J, Miñana G, Mainar L, Santas E, Merlos P, Rumiz E, Darmofal H, Heatta AM. Usefulness of the neutrophil to lymphocyte ratio in predicting long-term mortality in ST segment elevation myocardial infarction. The American journal of cardiology. 2008 Mar 15;101(6):747-52.
12. Tamhane UU, Aneja S, Montgomery D, Rogers EK, Eagle KA, Gurm HS. Association between admission neutrophil to lymphocyte ratio and outcomes in patients with acute coronary syndrome. The American journal of cardiology. 2008 Sep 15;102(6):653-7.
13. Azab B, Zaher M, Weiserbs KF, Torbey E, Lacossiere K, Gaddam S, Gobunsuy R, Jadonath S, Baldari D, McCord D, Lafferty J. Usefulness of neutrophil to lymphocyte ratio in predicting short-and long-term mortality after non–ST-elevation myocardial infarction. The American journal of cardiology. 2010 Aug 15;106(4):470-6.

14. Erikssen G, Liestøl K, Bjørnholt JV, Stormorken H, Thaulow E, Erikssen J. Erythrocyte sedimentation rate: a possible marker of atherosclerosis and a strong predictor of coronary heart disease mortality. European heart journal. 2000 Oct 1;21(19):1614-20.