DEVELOPMENTAL MEDICO-LIFE-SCIENCES

ISSN (P): 3007-2786, (E): 3007-2794 DOI: https://doi.org/10.69750/dmls.02.05.0130

ORIGINAL RESEARCH ARTICLE Open Access

Serum C-Reactive Protein and IL-6 Levels as Predictors of Disease Severity in Patients with Acute Myocardial Infarction

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ABSTRACT

Background: Inflammation during acute myocardial infarction (AMI) critically influences infarct size, ventricular remodeling, and subsequent clinical outcomes. Systemic inflammatory markers particularly interleukin-6 (IL-6) and high-sensitivity C-reactive protein (hs-CRP) are well established. However, their utility for forecasting early risk and disease severity in South Asian AMI patients remains unclear.

Objectives: To determine whether admission levels of hs-CRP and IL-6 individually predict AMI severity and whether a combined biomarker approach offers superior discrimination of high-risk clinical profiles.

Methods: From January to December 2023, we conducted a prospective observational study enrolling 150 patients aged 30–80 years who presented within 24 hours of symptom onset to two tertiary care centers in Lahore, Pakistan. Blood samples were obtained before reperfusion therapy to measure hs-CRP (immunoturbidimetric assay) and IL-6 (high-sensitivity ELISA). Severe AMI was defined as Killip class ≥ II, left ventricular ejection fraction < 40%, or GRACE score ≥ 156. Multivariable logistic regression identified independent predictors, and receiver operating characteristic (ROC) analyses determined optimal cut-offs and diagnostic accuracy.

Results: Of 150 patients, 58 (38.7%) met criteria for severe AMI. Severe cases had significantly higher median hs-CRP (14.8 mg/L vs 7.9 mg/L) and IL-6 (30.6 pg/mL vs 17.4 pg/mL) than non-severe cases (both $p < 0.001$). After adjustment, hs-CRP ≥ 10 mg/L (OR 3.12) and IL-6 ≥ 25 pg/mL (OR 4.28) independently predicted severity. The AUC was 0.82 for hs-CRP, 0.86 for IL-6, and 0.90 for the combined model, with sensitivity and specificity both >84%.

Conclusion: Elevated admission hs-CRP and IL-6 levels are independent, complementary predictors of AMI severity. A dual-marker model substantially improves early risk stratification and may support targeted monitoring and treatment in resource-limited settings.

Keywords: Acute Myocardial Infarction, High-Sensitivity C-Reactive Protein, Interleukin-6, Inflammation, Biomarker, Risk Stratification.

Received: 11/04/2025
Revised: 23/08/2025
Accepted: 27/08/2025
Published: 31/08/2025

INTRODUCTION

The degree of myocardial injury and the subsequent ventricular remodeling are strongly influenced by the intense inflammatory cascade triggered by acute myocardial infarction (AMI) [1]. For decades, C-reactive protein (CRP), an acute-phase protein synthesized by hepatocytes in response to pro-inflammatory cytokines, has been used as a prognostic marker in patients with acute coronary syndromes [2]. CRP levels rise within hours of myocardial necrosis, and early work by Morrow and colleagues demonstrated that patients with unstable angina or non-Q-wave myocardial infarction with elevated CRP at admission had significantly higher 14-day mortality [3]. More recently, Mitsis et al. confirmed the clinical and therapeutic importance of high-sensitivity CRP (hs-CRP), showing it to be a powerful predictor of in-hospital heart failure and long-term mortality in AMI patients [4]. Other studies and meta-analyses further emphasize the association of hs-CRP with adverse cardiovascular outcomes, underscoring its prognostic role [5,6].

Interleukin-6 (IL-6) is a multifunctional cytokine secreted by monocytes, vascular endothelial cells, and activated platelets at sites of vascular injury [7]. In contrast to CRP, which reflects a downstream hepatic response, IL-6 rises earlier often within a few hours of infarction and directly promotes endothelial activation, leukocyte adhesion, and prothrombotic signaling [8]. Mehta and collaborators demonstrated that admission IL-6 levels were closely associated with infarct size and Killip class, offering superior short-term prognostic accuracy compared with CRP alone [9]. Similarly, Bouzidi et al. identified an IL-6 threshold of 9.5 pg/mL as predictive of severe coronary artery disease in STEMI patients, highlighting its importance in early clinical risk assessment [10]. Evidence also suggests that IL-6 correlates with adverse events such as cardiogenic shock and ventricular dysfunction, further strengthening its value as a prognostic biomarker [11,12].

Although biologically interconnected, hs-CRP and IL-6 provide distinct yet complementary prognostic insights. In a systematic review, Zhang and colleagues demonstrated that adding hs-CRP to multivariable models improved prediction of in-hospital heart failure and all-cause mortality, while including IL-6 further enhanced risk discrimination [13]. Likewise, Held and colleagues reported that IL-6 independently predicted unfavorable cardiac outcomes, even after adjustment for hs-CRP and conventional cardiovascular risk factors [14]. This supports the growing evidence base that a dual-marker strategy may outperform single-marker models in risk stratification [15]. In South Asian populations, where genetic predisposition and inflammatory responses differ from Western cohorts, evidence on the combined predictive role of hs-CRP and IL-6 remains limited [16]. South Asians are disproportionately affected by premature coronary artery disease, and the American Heart Association has highlighted elevated inflammatory markers as a key contributor [17]. Establishing population-specific thresholds for hs-CRP and IL-6 could therefore improve early risk stratification and guide the efficient allocation of resources in overcrowded coronary care units across Pakistan and other low- and middle-income countries [18,19].

Based on these considerations, this prospective observational study was designed to evaluate admission levels of hs-CRP and IL-6 as individual and combined predictors of AMI severity in Pakistani patients. By identifying optimal cut-off values and assessing their incremental prognostic contribution beyond established risk scores, we aim to propose a practical biomarker-based framework for recognizing high-risk patients who may benefit

from closer monitoring and targeted therapeutic strategies [20,21].

MATERIALS AND METHODS

This prospective observational study was carried out from January to December 2023 at two tertiary cardiac institutions in Pakistan and all procedures followed the standards described in the Helsinki Declaration. Each subject provided written informed permission before to participation. Eligible participants were people aged 30 to 80 who came within 24 hours of experiencing chest pain and satisfied the diagnostic criteria for type-1 acute myocardial infarction. Ischemic chest pain, increased cardiac troponin I over the 99th percentile threshold, and either new ST-segment elevation, dynamic ST-T alterations on serial electrocardiograms, or a new left bundle branch block were necessary for the diagnosis.

Patients were excluded if they had disorders that may interfere with inflammatory biomarker tests. These included continuing infections, autoimmune or chronic inflammatory illnesses, cancer, significant surgery or trauma within the previous month, severe renal or hepatic dysfunction, and the use of corticosteroids or other immunomodulatory medications. At admission, demographic information (age, gender), cardiovascular risk factors (hypertension, diabetes, smoking status), and infarction type (STEMI or NSTEMI) were recorded. The symptom-to-door time was recorded. Killip classification was used to determine clinical severity during a physical examination. Within 72 hours of hospitalization, experienced cardiologists performed transthoracic echocardiography using the biplane Simpson's technique to determine the left ventricular ejection fraction (LVEF). Each patient's Global Registry of Acute Coronary Events (GRACE) score was also computed. Disease severity was determined by the following criteria: Killip class II or higher, LVEF < 40%, or GRACE score ≥156. Peripheral venous blood was drawn before reperfusion therapy or anti-inflammatory drugs were administered. After being collected in plain tubes, the samples were centrifuged at 3,000 × g for 10 minutes after being allowed to clot for 30 minutes at room temperature. Before analysis, serum aliquots were stored in a refrigerator at -80 degrees Celsius.

An immunoturbidimetric assay on the Roche Cobas 8000 platform was used to assess high-sensitivity CRP (hs-CRP), which has an analytical sensitivity of 0.15 mg/L and an intra-/inter-assay variability of less than 5%. Interleukin-6 levels were measured using a high-sensitivity sandwich ELISA (Human IL-6 Quantikine HS, R&D Systems) with a detection threshold of 0.11 pg/mL and intra-/inter-assay variability of less than 6%. Laboratory experts who were not informed of the clinical outcomes performed each analysis in triplicate. SPSS version 29 was used to process the data. The Shapiro-Wilk test was used to assess the distribution of continuous data. If appropriate, the results were displayed as the median with interquartile range or mean ± standard deviation.

The Student's t-test or Mann-Whitney U test were used to compare continuous variables, while the χ^2 test or Fisher's exact test was used to compare categorical variables. Spearman's rank correlation was used to examine the relationships among hs-CRP, IL-6, LVEF, and peak troponin I. To find independent predictors of severe AMI, including covariates with $p < 0.10$ in univariate analysis, logistic regression models were employed. Youden's index was utilized to establish suitable thresholds, and receiver operating characteristic (ROC) curves were developed to assess the discriminatory performance of hs-CRP, IL-6, and their combination. .A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

Between January and December 2023, 150 patients diagnosed with acute myocardial infarction (AMI) were enrolled in the study. The cohort included 113 men (75.3%) and 37 women (24.7%), with a mean age of 58.2 ± 9.7 years. Severe AMI defined as Killip class ≥ II, left ventricular ejection fraction (LVEF) < 40%, or a GRACE score > 156 was identified in 58 patients (38.7%), including 45 men and 13 women. There were no significant differences in baseline cardiovascular risk factors between the severe and non-severe groups when stratified by gender. These risk factors included dyslipidemia, diabetes mellitus, hypertension, and family history of coronary artery disease. However, patients in the severe AMI group presented to the hospital significantly later after the onset of chest pain compared with the non-severe group (6.1 ± 2.4 hours vs. 4.8 ± 1.9 hours; p = 0.003). This trend was observed in both men and women (Table 1).

Table-1: Baseline Demographics and Clinical Characteristics by Sex and Severity (N = 150)

Variable	Overall (n=150)	Men (n=113)	Women (n=37)	p-value*
		Non-severe (68) / Severe (45)	Non-severe (24) / Severe (13)	
Age, years mean ± SD	58.2 ± 9.7	57.6 ± 9.8 / 59.1 ± 10.0	60.3 ± 9.1 / 60.2 ± 10.4	0.27
Symptom-to-door time, h mean ± SD	5.3 ± 2.2	4.9 ± 1.9 / 6.0 ± 2.3	4.5 ± 1.8 / 6.4 ± 2.6	0.003
STEMI presentation n (%)	112 (74.7)	48 / 38 (70.6 % / 84.4 %)	18 / 8 (75.0 % / 61.5 %)	0.30
Hypertension n (%)	93 (62.0)	42 / 30 (61.8 % / 66.7 %)	13 / 8 (54.2 % / 61.5 %)	0.48
Diabetes mellitus n (%)	70 (46.7)	31 / 25 (45.6 % / 55.6 %)	10 / 4 (41.7 % / 30.8 %)	0.52
Current smoking n (%)	49 (32.7)	29 / 20 (42.6 % / 44.4 %)	0 / 0 (0 % / 0 %)	0.72
Dyslipidemia n (%)	58 (38.7)	27 / 20 (39.7 % / 44.4 %)	8 / 3 (33.3 % / 23.1 %)	0.83
Family history of CAD n (%)	44 (29.3)	19 / 14 (27.9 % / 31.1 %)	7 / 4 (29.2 % / 30.8 %)	0.71
Killip class ≥ II n (%)	58 (38.7)	/ 45	/ 13	
LVEF, % mean ± SD	45.8 ± 8.7	49.2 ± 5.5 / 40.3 ± 7.9	50.1 ± 4.7 / 37.9 ± 8.4	<0.001
GRACE score median (IQR)	148 (132–162)	136 (122–148) / 164 (159–183)	142 (127–150) / 174 (162–186)	<0.001

**Overall comparison between severe and non-severe groups.*

In admission samples, inflammatory biomarkers were significantly higher in the severe AMI group. The median high-sensitivity CRP (hs-CRP) in severe cases was 14.8 mg/L (IQR 10.5-20.9), compared to 7.9 mg/L (IQR 5.2-11.6) in non-severe cases (p < 0.001). The median interleukin-6 (IL-6) level was 30.6 pg/mL (IQR 23.9-38.2), compared to 17.4 pg/mL (IQR 12.1-22.8) (p < 0.001). Both men and women followed these patterns, with no significant sex differences (Table 2). Severity was associated with peak troponin-I release (18.3 ± 8.2 ng/mL vs. 12.8 ± 6.5 ng/mL; p < 0.001), and echocardiography revealed poorer LVEF and higher end-diastolic diameter in severe instances.

Table-2: Admission Biomarkers and Echocardiographic Measures by Severity

Parameter	Non-severe (n=92)	Severe (n=58)	p-value
hs-CRP, mg/L median (IQR)	7.9 (5.2–11.6)	14.8 (10.5–20.9)	<0.001
IL-6, pg/mL median (IQR)	17.4 (12.1–22.8)	30.6 (23.9–38.2)	<0.001
Peak troponin-I, ng/mL mean ± SD	12.8 ± 6.5	18.3 ± 8.2	<0.001
LVEF, % mean ± SD	49.5 ± 5.2	39.8 ± 8.1	<0.001
LV end-diastolic diameter, mm	50.2 ± 4.1	54.7 ± 5.8	<0.001

After adjusting for age, gender, STEMI presentation, diabetes, and symptom-to-door time, hs-CRP ≥ 10 mg/L (adjusted OR 3.12; 95% CI 1.47-6.39; p = 0.002) and IL-6 ≥ 25 pg/mL (adjusted OR 4.28; 95% CI 2.03-9.04; p < 0.001) were independent predictors of severe AMI (Table 3). A symptom-to-door period of more than 6 hours independently enhanced the likelihood of severity (adjusted OR 2.05; 95% CI 1.01-4.16; p = 0.048).

Table-3: Multivariable Predictors of Severe AMI

Predictor	Adjusted OR	95 % CI	p-value
hs-CRP ≥ 10 mg/L	3.12	1.47–6.39	0.002
IL-6 ≥ 25 pg/mL	4.28	2.03–9.04	<0.001
Symptom-to-door ≥ 6 h	2.05	1.01–4.16	0.048
STEMI presentation	1.32	0.62–2.82	0.47
Diabetes mellitus	1.18	0.56–2.49	0.66

The receiver-operating characteristic analysis (Table 4) demonstrated strong discrimination for both markers: hs-CRP AUC = 0.82 (95% CI 0.75-0.89) and IL-6 AUC = 0.86 (95% CI 0.80-0.92). The optimal cut-offs (hs-CRP 10.2 mg/L; IL-6 24.8 pg/mL) resulted in sensitivities of 72.4% and 79.3%, respectively, and specificities of 80.4% and 81.5%. Combining both markers increased the AUC to 0.90 (95% CI 0.85-0.95), with 84.5% sensitivity and 85.9% specificity.

Table-4: Diagnostic Performance of hs-CRP and IL-6 for Severe AMI

Marker	AUC	95 % CI	Cut-off	Sensitivity (%)	Specificity (%)
hs-CRP	0.82	0.75–0.89	10.2 mg/L	72.4	80.4
IL-6	0.86	0.80–0.92	24.8 pg/mL	79.3	81.5
hs-CRP + IL-6 combined	0.90	0.85–0.95		84.5	85.9

Patients with severe AMI had considerably greater inflammatory markers and worse echocardiographic results than non-severe instances. Both hs-CRP and IL-6 independently predicted clinical severity, with IL-6 having significantly better discrimination. The dual-marker strategy improved risk stratification even more, indicating that measuring hs-CRP and IL-6 on admission can effectively identify high-risk AMI patients, regardless of gender, who may benefit from increased surveillance and early management.

DISCUSSION

High-sensitivity C-reactive protein (hs-CRP) and interleukin-6 (IL-6) admission concentrations were found to be strongly associated with markers of severe disease, such as elevated Killip class, reduced left ventricular ejection fraction (LVEF), and higher GRACE scores, in this prospective cohort of acute myocardial infarction (AMI) patients from Pakistan. The risk of severe AMI was nearly three times higher for individuals with hs-CRP levels above 10 mg/L and almost four times higher for those with IL-6 levels above 25 pg/mL. These associations remained significant even after adjusting for time to hospital arrival and conventional cardiovascular risk factors. Importantly, combining both biomarkers yielded an AUC of 0.90, demonstrating strong discriminative power for identifying high-risk presentations [18].

These observations are consistent with prior studies linking systemic inflammatory

markers to adverse outcomes following AMI. Early work by Morrow and colleagues showed that CRP levels at admission predicted short-term mortality in patients with acute coronary syndromes [19], and subsequent investigations confirmed associations with infarct size, ventricular dysfunction, and poor prognosis [20,21]. Likewise, Mehta et al. highlighted the rapid rise of IL-6 after myocardial injury, showing that it correlated more strongly than CRP with infarct burden and the risk of cardiogenic shock [22,23].

The present study extends these findings by directly evaluating the predictive performance of both markers in a South Asian population, which has historically been underrepresented in cardiovascular biomarker study. Our results indicate that IL-6 may offer greater sensitivity and specificity than hs-CRP alone; however, their combined use substantially improves predictive accuracy. This aligns with evidence from Zhang and colleagues, who demonstrated that adding IL-6 to CRP-based models significantly enhanced prognostic value [24,25].

The pathophysiological rationale for these observations lies in the sequential activation of the inflammatory cascade following AMI. IL-6 is released by ischemic myocardium and activated leukocytes within hours, stimulating hepatic CRP synthesis and promoting further leukocyte recruitment [26,27]. Elevated IL-6 levels may therefore reflect both the magnitude of initial myocardial injury and the extent of systemic inflammation, whereas CRP provides a downstream measure of hepatic response. Measuring both biomarkers captures complementary aspects of the inflammatory process, thus improving early risk stratification, especially in resource-limited settings where advanced imaging or prolonged monitoring is not always feasible [28–30].

Several limitations merit consideration. First, the single-center design in Lahore may limit generalizability to broader South Asian or global populations with different genetic and environmental characteristics [31]. Second, biomarker measurements were restricted to admission samples; serial measurements could have provided insights into the temporal dynamics of inflammation and its relationship with remodeling or long-term outcomes [32]. Third, although key covariates were adjusted for, residual confounding from unmeasured variables such as infarct size assessed by cardiac MRI or use of anti-inflammatory drugs cannot be excluded [33]. Finally, the sample size, while adequate to detect biomarker differences, may have been insufficient for robust subgroup analyses, including sex-specific thresholds [34].

Despite these limitations, our study has several strengths. We recruited a well-defined AMI population with standardized echocardiographic assessment and GRACE scoring, ensuring objective classification of severity [35]. Biomarker assays were performed using high-sensitivity techniques with minimal analytical variability, and laboratory staff were blinded to clinical outcomes [36]. Most importantly, by establishing context-specific cut-offs for hs-CRP and IL-6, we provide actionable thresholds that may be directly applicable in Pakistani and other resource-constrained coronary care units [37–43].

CONCLUSION

Admission concentrations of hs-CRP and IL-6 serve as independent yet complementary predictors of early severity in acute myocardial infarction (AMI). Utilizing both biomarkers together specifically, hs-CRP values above 10 mg/L and IL-6 levels ≥ 25 pg/mL provides superior risk stratification compared with either marker individually, and may support more targeted monitoring and therapeutic decision-making in settings with limited resources. To

strengthen these findings, future large-scale, multicenter investigations are warranted to validate these thresholds and to explore whether biomarker-driven strategies can translate into improved patient outcomes.

Conflict of Interest:

The authors report no conflicts of interest.

Funding:

No external funding was received for this study.

Acknowledgments:

We gratefully acknowledge our colleagues and all study participants for their valuable contribution.

Authors' contributions:

EA and TA conceived and designed the study. IR and IG collected the data. FAR and TA performed laboratory analyses. EA and IR conducted statistical analysis and interpretation. EA and IG drafted the manuscript. All authors critically revised the manuscript, approved the final version, and agreed to be accountable for all aspects of the work.

Data Availability Statement:

The data used in this study are available upon reasonable request from the corresponding author, subject to ethical and institutional guidelines.

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This Article May be cited As: *Asghar E, Aziz T, Ramzan I, Gul I, Razzaq FA. Serum C-Reactive Protein and IL-6 Levels as Predictors of Disease Severity in Patients with Acute Myocardial Infarction: CRP and IL-6 as Predictors of AMI Severity. DEVELOPMENTAL MEDICO-LIFE-SCIENCES. 2025;2(5):44-52.doi: 10.69750/dmls.02.05.0130*