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A Comprehensive Analysis of Clinical and Laboratory Features in Patients with Acute Coronary Syndrome and Associated Inflammatory Syndrome

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ABSTRACT

Background: Acute coronary syndrome (ACS) is a significant contributor to cardiovascular morbidity and mortality, with inflammation playing a critical role in its development and progression. C-reactive protein (CRP) is an extensively studied inflammatory biomarker in cardiovascular research. This study aims to analyze the clinical and laboratory features of ACS patients in relation to their inflammatory status, as expressed by high-sensitivity CRP (hs-CRP) levels. **Methods:** This prospective observational study included 159 patients who underwent percutaneous coronary intervention for acute myocardial infarction. Patients were divided into two groups based on hs-CRP levels: Group 1 (hs-CRP ≥ 3 mg/dL) and Group 2 (hs-CRP < 3 mg/dL). Demographic, clinical, laboratory, and angiographic data were collected and analyzed. **Results:** Patients with elevated hs-CRP levels were significantly older ($p = 0.02$) and exhibited higher peak troponin I levels ($p < 0.001$) and lower left ventricular ejection fraction ($p = 0.003$). The number of stents implanted was higher in the elevated hs-CRP group ($p = 0.008$). In-hospital mortality was significantly higher in the group with elevated hs-CRP levels ($p = 0.04$). **Conclusion:** Elevated hs-CRP levels in ACS patients are associated with greater myocardial injury, reduced cardiac function, and increased in-hospital mortality. These results reinforce the importance of inflammation in the pathophysiology and prognosis of ACS and underscore the potential utility of hs-CRP as a prognostic biomarker. Future large-scale, multicenter studies are warranted to validate these findings and explore targeted anti-inflammatory therapies in improving clinical outcomes.

Keywords: acute coronary syndrome, high-sensitivity c-reactive protein, inflammation, prognostic biomarker, myocardial injury

INTRODUCTION

Acute coronary syndrome (ACS) represents a major category of cardiovascular pathology and remains a leading contributor to morbidity and mortality worldwide.[1] Within this clinical spectrum, acute myocardial infarction (AMI) is diagnosed when there is definitive evidence of myocardial cell death, or necrosis, attributable to ischemic injury resulting from an acute and critical reduction, or complete cessation, of coronary blood flow.[2]

The conventional diagnostic framework for AMI delineates two primary subtypes: ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), with differentiation based on the presence or absence of persistent ST-segment elevation on a standard 12-lead ECG. Persistent ST-segment elevation typically reflects transmural myocardial injury and is often associated with a more urgent need for reperfusion therapy. Conversely, NSTEMI lacks this ECG finding but nonetheless represents a clinically significant acute coronary event requiring timely intervention. Despite this ECG-based dichotomy, STEMI and NSTEMI share a common pathophysiological substrate. In most cases, both arise from the acute rupture or erosion of a vulnerable atherosclerotic plaque within the coronary arteries. This event precipitates the rapid development of an occlusive or subocclusive thrombus, which critically impairs myocardial perfusion. The ensuing oxygen supply–demand mismatch initiates a cascade of cellular injury, culminating in irreversible myocardial necrosis if reperfusion is not promptly achieved.[3]

Myocardial infarction (MI) represents one of the most severe and potentially fatal manifestations of coronary artery disease (CAD) and is a major precipitating factor for sudden cardiac death (SCD).[4] Epidemiological data indicate that each year more than 3 million individuals experience STEMI, while over 4 million are affected by NSTEMI. Although historically MI has been more frequently reported in high-income, industrialized nations, its prevalence in low- and middle-income countries has risen considerably in recent decades, reflecting a globalizing burden of atherothrombotic disease.[5] Despite significant advances in diagnostic strategies, pharmacological therapies, and revascularization techniques, MI remains a leading cause of death worldwide.

Inflammation plays a central role in the initiation, progression, and clinical manifestation of atherosclerosis, serving as a fundamental pathophysiological mechanism underlying CAD. Over the past decade, there has been a growing research emphasis on the identification and clinical utility of inflammatory biomarkers as tools for risk stratification, diagnosis, and prognosis in patients with CAD. These biomarkers not only provide insight into the underlying disease activity but may also offer predictive value in anticipating acute cardiovascular events. The destabilization and subsequent rupture of atherosclerotic plaques, events that precipitate ACS, are strongly linked to heightened inflammatory activity within the vascular wall. This inflammatory state contributes to the weakening of the fibrous cap, increased lipid core vulnerability, and promotion of thrombus formation, collectively driving the transition from a stable to an unstable clinical state. Understanding and monitoring these inflammatory processes have, therefore, become critical components of modern cardiovascular research and patient management.[6]

C-reactive protein (CRP) is one of the most extensively investigated inflammatory biomarkers in cardiovascular research and remains extremely important in the search for reliable predictors of CHD risk. Unlike many other markers of systemic inflammation, high-sensitivity CRP (hs-CRP) assays enable accurate, reproducible, and cost-effective measurement, making them suitable for both large-scale epidemiologic studies and clinical risk assessment. Beyond its role as a marker, accumulating evidence suggests that CRP may exert direct pathogenic effects in the development and progression of atherosclerosis, underscoring its dual significance: not only as a biomarker of vascular inflammation but also as a potential mediator of CAD pathophysiology.[7] Due to its stability in circulation, minimal daily fluctuation, and the availability of sensitive, affordable assays, CRP measurement has become a cornerstone in evaluating inflammatory status in cardiovascular patients.

In the setting of AMI, CRP levels rise swiftly following myocardial cell death, typically peaking within 1 to 3 days after symptom onset. Higher CRP values correlate with larger infarct size and a greater inflammatory response, serving as a prognostic marker for complications such as heart failure, ventricular arrhythmias, cardiogenic shock, and increased mortality.[8] CRP is also involved in the pathological processes of atherosclerosis: it modulates endothelial function by downregulating nitric oxide synthase, enhances macrophage uptake of LDL cholesterol, and promotes endothelial adhesion molecule expression, thereby actively contributing to vascular inflammation and plaque instability. The prognostic implications of CRP extend beyond the acute phase; persistent elevation following AMI predicts adverse cardiac remodeling and the development of chronic heart failure, reinforcing its significance in guiding both immediate and long-term therapeutic strategies.[9]

The aim of this study was to analyze the clinical and laboratory features of patients with ACS and associated inflammatory syndrome, expressed by elevated hs-CRP levels.

MATERIALS AND METHODS

This prospective observational study was conducted on 159 patients who underwent percutaneous coronary intervention for AMI in the Cardiology Department of the County Emergency Clinical Hospital of Târgu Mureș between November 2024 and June 2025. The cohort comprised 107 patients with STEMI and 52 with NSTEMI. Patients with any concurrent infection at admission were excluded, given its potential impact on systemic inflammation. Those with known chronic inflammatory disorders were also excluded.

Demographic and clinical variables were collected for each patient at admission. Inflammatory status was assessed by measuring hs-CRP levels. Cardiac function was evaluated by left ventricular ejection fraction (LVEF) using echocardiography. Coronary angiography data included the number of stents placed, the affected coronary artery, and the presence of residual lesions after intervention. Therapeutic measures during hospitalization were also recorded.

Based on inflammatory status, patients were divided into two groups: Group 1, with hs-CRP ≥ 3 mg/dl (n = 86), and Group 2, with hs-CRP < 3 mg/dl (n = 73).

Statistical analysis was performed using GraphPad Prism v.8.0 (GraphPad Software). Results are expressed as mean ± s.d. for continuous variables, and categorical data are presented as absolute numbers and percentages. Parametric tests were used to compare continuous variables, and the Chi-square test was applied for categorical variables. A p value < 0.05 was considered statistically significant.

Ethical approval for the study was obtained from the institutional ethics committee prior to data collection (approval no. 12250/28.05.2024).

RESULTS

The demographic and clinical characteristics of patients with ACS were analyzed according to their hs-CRP levels (Table 1). Patients with elevated hs-CRP levels (≥ 3 mg/dl) were significantly older than those with lower hs-CRP levels (< 3 mg/dl). The proportion of male patients was similar between the two groups. No significant differences were found in the prevalence of diabetes mellitus, hypertension, smoking status, or history of prior myocardial infarction.

Laboratory and angiographic findings are presented in Table 2. Patients with elevated hs-CRP levels (≥ 3 mg/dl) had significantly higher hs-CRP concentrations, confirming the inflammatory status of this group. They also exhibited significantly higher peak troponin I levels, indicating greater myocardial injury, and lower left ventricular ejection fraction (LVEF), suggesting impaired cardiac function. Furthermore, a greater number of stents were implanted in this group, which may reflect more extensive CAD or more complex interventions. The distribution of affected coronary arteries was similar between the two

TABLE 1. Demographic and clinical characteristics of patients with ACS according to hs-CRP levels

Variable	Group 1 (n = 86)	Group 2 (n = 73)	p value
Age (years, mean ± s.d.)	62.3 ± 10.5	58.4 ± 9.8	0.02
Male sex, n (%)	65 (75.6%)	55 (75.3%)	0.95
Diabetes mellitus, n (%)	38 (44.2%)	24 (32.9%)	0.09
Hypertension, n (%)	55 (63.9%)	47 (64.4%)	0.93
Smoking, n (%)	48 (55.8%)	39 (53.4%)	0.72
Previous MI, n (%)	12 (14.0%)	8 (11.0%)	0.49

TABLE 2. Laboratory and angiographical findings

Variable	Group 1 (n = 86)	Group 2 (n = 73)	p value
hs-CRP (mg/dl)	4.8 ± 1.2	1.8 ± 0.6	<0.001
LVEF (%)	45.2 ± 7.8	48.7 ± 6.5	0.003
Peak troponin I (ng/ml)	35.2 ± 10.4	22.5 ± 8.9	<0.001
Number of stents implanted	1.4 ± 0.5	1.2 ± 0.4	0.008

TABLE 3. Clinical outcomes and hospital data

Variable	Group 1 (n = 86)	Group 2 (n = 73)	p value
Heart failure on admission, n (%)	18 (20.9%)	9 (12.3%)	0.12
Recurrent ischemia, n (%)	11 (12.8%)	6 (8.2%)	0.33
In-hospital mortality, n (%)	5 (5.8%)	1 (1.4%)	0.04

groups, with the anterior descending artery (LAD), right coronary artery (RCA), and left circumflex artery (LCx) involved in comparable proportions (p = 0.62), indicating no significant difference in the location of disease according to hs-CRP levels. Overall, these findings suggest that higher hs-CRP levels are associated with greater myocardial injury and reduced cardiac function, but not necessarily with differences in the distribution of coronary artery involvement.

Although the incidence of heart failure at admission was higher in the group with elevated hs-CRP, this difference did not reach statistical significance. Similarly, recurrent ischemia during hospitalization was slightly more frequent in the high hs-CRP group, but without a significant difference. In contrast, the in-hospital mortality rate was significantly higher among patients with elevated hs-CRP compared to those with lower levels (Table 3). This suggests that elevated hs-CRP is associated with an increased risk of mortality during hospitalization for ACS. Overall, while adverse events such as heart failure and recurrent ischemia showed trends toward higher rates in the high hs-CRP group, only the difference in in-hospital mortality was statistically significant, indicating a potential prognostic value of hs-CRP in predicting mortality outcomes during the acute phase of treatment.

DISCUSSION

This study aimed to elucidate the clinical and laboratory characteristics of patients with ACS in relation to their inflammatory status, as reflected by hs-CRP levels. The findings highlight several important aspects: elevated hs-CRP levels are associated with greater myocardial injury, impaired cardiac function, and increased in-hospital mortality, consistent with a growing body of evidence linking systemic inflammation to adverse cardiovascular outcomes.

Our data demonstrated that patients with higher hs-CRP levels were significantly older, consistent with previous research suggesting that aging is associated with chronic low-grade inflammation, often termed 'inflammaging', which may accelerate atherosclerotic progression and instability.[10,11] Despite similar prevalence of common risk factors such as hypertension, diabetes, and smoking across both groups, patients with elevated hs-CRP exhibited greater myocardial damage, reflected by higher peak troponin I levels, and worse cardiac function, as indicated by lower LVEF. These results are in accordance with earlier studies that identified hs-CRP as a marker of infarct size and myocardial damage.[12,13]

The angiographic findings revealed no significant difference in the distribution of affected coronary arteries between groups, indicating that inflammatory status may not directly influence the location of coronary lesions but rather their destabilization and propensity to cause infarction. Interestingly, patients with higher hs-CRP required slightly more stents, possibly reflecting more complex or extensive coronary disease. This observation supports prior evidence linking systemic inflammation with greater coronary plaque burden and complexity.[14,15]

One of the most notable results of this study is the significantly higher in-hospital mortality among patients with elevated hs-CRP. Similar associations have been described in previous research, which identified hs-CRP as an independent predictor of short-term mortality post-AMI.[16,17] Elevated inflammatory markers, including hs-CRP, are believed to contribute to a pro-thrombotic and pro-inflammatory milieu that exacerbates myocardial injury and impairs recovery.[18] Furthermore, the trend towards higher rates of heart failure and recurrent ischemia, although not statistically significant, underscores the potential role of hs-CRP as a risk stratification tool for adverse outcomes.

Several studies have emphasized the prognostic value of hs-CRP in patients with ACS. For instance, the CRP in Acute Myocardial Infarction Trial (CRP-AMI) showed that elevated hs-CRP levels early after MI predict long-term adverse events, including mortality.[19] Additionally, meta-analyses have confirmed the role of hs-CRP as a reliable biomarker for predicting not only short-term mortality but also future cardiovascular events.[20]

The mechanistic link between hs-CRP and adverse outcomes remains an active area of investigation. Beyond serving as an inflammatory marker, experimental data suggest that CRP may actively participate in atherothrombosis

by inducing endothelial dysfunction, promoting smooth muscle cell proliferation, and increasing the expression of adhesion molecules, which can destabilize atherosclerotic plaques.[21,22] These effects may help explain the observed correlation between elevated hs-CRP levels and worse clinical outcomes.

The clinical implications of our findings support integrating hs-CRP measurement into the routine assessment of patients with ACS. Identifying high-risk individuals based on inflammatory markers can optimize stratification and guide therapeutic approaches, including anti-inflammatory strategies. Recent trials, such as the CANTOS trial, have demonstrated that targeted anti-inflammatory therapy reduces recurrent cardiovascular events, further validating the pathogenic role of inflammation in atherosclerosis.[23]

Nevertheless, it is important to note the limitations of this study. The observational design precludes establishing causality, and although exclusion criteria were applied, potential confounders influencing hs-CRP levels, such as subclinical infections or other inflammatory conditions, cannot be entirely ruled out. In addition, the relatively small sample size and single-center setting may restrict the generalizability of these findings.

CONCLUSION

Our study demonstrates that elevated hs-CRP levels in ACS patients are associated with greater myocardial injury, reduced cardiac function, and increased in-hospital mortality. These findings reinforce the central role of inflammation in the pathophysiology and prognosis of ACS and highlight the potential of hs-CRP as a prognostic biomarker. Future large-scale, multicenter studies are needed to validate these results and to assess whether targeted anti-inflammatory therapies can improve clinical outcomes.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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