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Relationship between Systemic Inflammatory Biomarkers and LAD Plaque Lesion Localization and Severity in Patients with Acute ST-segment Elevation Myocardial Infarction

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ABSTRACT

Introduction: Acute ST-segment elevation myocardial infarction (STEMI) is a leading cause of mortality, driven by coronary artery disease and inflammation. Systemic inflammatory biomarkers, including neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and C-reactive protein-to-albumin ratio (CAR), are linked to lesion severity and localization in the left anterior descending (LAD) artery, but local data from Pakistan are scarce. **Objective:** To assess the relationship between systemic inflammatory biomarkers (NLR, PLR, CAR) and the localization and severity of LAD plaque lesions in STEMI patients. **Material and Methods:** This retrospective study at Dr. Ruth K.M. Pfau Civil Hospital, Karachi from April, 2024 to September, 2024, included 87 STEMI patients with proximal or mid-LAD lesions. Biomarkers were correlated with angiographic findings using SPSS version 23.0. **Results:** Proximal LAD lesions (N=53) showed higher median NLR (3.00 vs. 1.50), PLR (109.75 vs. 70.00), and CAR (35.71 vs. 14.00) compared to mid-LAD lesions (N=33), indicating greater inflammatory burden ($p<0.05$). **Conclusion:** Elevated NLR, PLR, and CAR are associated with proximal LAD lesions, offering cost-effective risk stratification tools for STEMI patients in resource-limited settings.

INTRODUCTION

Acute coronary syndrome (ACS) is a group of clinical manifestations that are connected with myocardial ischemia, including unstable angina, ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) (1,2). The impact of coronary heart disease on a global level is excessive (3). Coronary heart disease is the most frequent cause of death in the world, as 3.8 and 3.4 million die each year of heart disease in men and women, respectively, regardless of geography or socioeconomic status divisions (4). Coronary heart disease deaths are significantly high among South Indians, especially those in low and medium-income countries such as Pakistan, as compared to other parts (5).

In Pakistan, a low- and middle-income nation in South Asia, coronary heart disease contributes to a substantial health burden, and its prevalence is worsened by individual demographic and risk factors (6). In the past, hyperlipidemia and low-density lipoprotein (LDL) deposition were seen as the key factors that cause stenosis in the coronary artery. Nonetheless, recent studies have changed this opinion, as inflammation has been seen as an important factor leading to the development and progression of atherosclerotic CAD and subsequent ACS (7). Significant over expression of the inflammatory markers as well as their related products has been related with greater severity of coronary artery disease in patients subjected to the percutaneous coronary intervention, emphasizing the role of systemic inflammation in ACS (8). Moreover, inflammation causes plaque instability, whereby the initiation of thrombus development and the ischemia-reperfusion injury have been demonstrated to exacerbate clinical outcomes of STEMI patients (9).

The use of systemic inflammatory biomarkers in the prognosis of ACS has raised interest (10). Routine blood-derived biomarkers are affordable and readily applicable in the clinics, particularly in low-resource countries such

as Pakistan (11). As an example, the systemic inflammatory immunity index was found to be relative to intracoronary thrombus burden in STEMI patients, being able to help stratify the risk (12). Likewise, new biomarkers such as CRTP5 and systemic immune-inflammation index showed a predictive value with respect to the severity of coronary artery disease and fibrosis of the myocardium in patients with non-ST-elevation ACS, which confirms their clinical significance (13).

Being beyond prognosis, inflammatory biomarkers can also become evidence of the localization of coronary lesions. The left anterior descending (LAD) artery, which is frequently implicated in the cases of STEMI, feeds a larger percentage of the myocardium, and lesions involving proximal or even middle areas of the artery are accompanied by an increased amount of damage to the myocardium (14). The NLR and CAR were also found to be higher in proximal LAD lesions in relation to mid-LAD lesions, known to have a worse prognosis and large infarct size (15). Such conclusions are confirmed by modern imaging methods that demonstrate an association of inflammatory biomarkers, macrophage influx, and plaque vulnerability (16). The manifold interaction of systemic inflammation and local coronary pathology, thrombus burden, and plaque features further challenges the necessity to investigate these relations of specific groups (17).

Notwithstanding the increasingly large body of evidence, local data on the utility of systemic inflammatory biomarkers in detecting the location and severity of LAD lesions in STEMI have yet to be obtained in Pakistan. This gap remains essential because the prevalence rates of coronary heart disease are high in South Asia, and potential biomarkers such as NLR, PLR, and CAR can better stratify risks in a low-resource context (18). Moreover, new biomarkers, including the uric acid to albumin ratio, have proved to be useful when predicting the outcome of non-ST-elevation myocardial infarction, signaling a wider use of inflammatory markers in the management of ACS (19). The results of this research may also be of high relevance to the practice of medicine, especially in low-resource settings where sophisticated diagnostic equipment is not readily available. With the help of readily available biomarkers, doctors can more accurately predict patients who are at risk of severe lesions on the LAD and adverse outcomes and mechanical interventions can be performed early enough to prevent events like heart failure, arrhythmias or sudden cardiac death. The study can also be used to generate knowledge about the role of inflammation in ACS, in correlating with the growing evidence on the role of systemic inflammatory markers in the pathogenesis and prognosis of coronary heart artery disease. It is hoped that through this retrospective study, practical data can be given that can be used to improve the quality of care that can be offered to STEMI patients within Pakistan and other comparable regions, and ultimately, decrease the burden of coronary heart disease on high-risk groups.

Objective

To evaluate the relationship between systemic inflammatory biomarkers (NLR, PLR, CAR) and the localization and severity of LAD plaque lesions in patients with acute ST-segment elevation myocardial infarction.

MATERIALS AND METHODS

Study Design: Retrospective Observational Cohort study.

Study Setting: The study was conducted at the Cardiology Unit of Dr. Ruth K.M. Pfau Civil Hospital, Karachi, Pakistan, using data retrieved from the catheterization laboratory and patient discharge files.

Duration of study: Data collection spanned from April 2024 to September 2024, covering records of STEMI patients admitted during this period.

Inclusion criteria: The eligible potential patients were to be aged over 18 and had enrolled with a diagnosis of acute anterior STEMI on the basis of the new left bundle branch block or ST-elevation in the anterior leads and chest pain lasting more than 20 minutes, and single-vessel disease in the proximal or middle left anterior descending (LAD) artery as documented by angiography.

Exclusion Criteria: Patients below 18 years, participants with acute pregnancies, lactating ladies, people who had received fibrinolytic therapy, and patients who suffered lesions in the right coronary and the left circumflex arteries were excluded. Besides, the distal LAD lesions, those patients with present coronary artery disease, trauma, surgery, neoplasm or those under immunosuppressive medication were not included.

Methods

The given retrospective study had its data collection carried out through discharge records with the patients visiting the Cardiology Unit of Dr. Ruth K.M. Pfau Civil Hospital, Karachi, in April to September of 2024, having acute (STEMI). The patients were selected based on the purposive sampling that involved non-probability sampling of patients who had angiographic evidence of single-vessel disease, proximal or middle left anterior descending (LAD) artery. The data consisted of demographic data (age, sex, smoking status, coronary risk factors), data obtained by laboratory tests (complete blood count, C-reactive protein, serum albumin, troponin I, renal function tests), echocardiographic data (left ventricular ejection fraction), and angiography reports. The systemic inflammatory markers were estimated in the form of neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and C-reactive protein-to-albumin ratio (CAR), which were correlated to the localization and severity of the LAD lesions. The analysis of data was conducted in SPSS version 23.0, and medians and interquartiles were presented as measures of continuous variables, and frequencies were presented as categorical variables, and associations were conducted using Chi-square, Fisher's exact, or Spearman, with $p<0.05$ as significant.

RESULTS

The study included 87 patients with acute ST-segment elevation myocardial infarction (STEMI) who underwent primary percutaneous coronary intervention (PCI) at Dr.

Ruth K.M. Pfau Civil Hospital, Karachi, from April 2024 to September 2024. The cohort was divided based on left anterior descending (LAD) artery lesion localization: 53 patients (60.9%) had proximal LAD involvement, and 33 patients (37.9%) had mid-LAD involvement. One of our patients had involvement of proximal to mid LAD. Demographic and clinical characteristics revealed distinct patterns between the groups, with systemic inflammatory biomarkers showing significant associations with lesion location and severity.

Table 1

Demographic and Clinical Characteristics

Characteristic	Proximal LAD (N=53)	Mid-LAD (N=33)
Males	39 (73.6%)	26 (78.8%)
Females	15 (28.3%)	7 (21.2%)
Smoking Status	31 (58.5%)	13 (39.4%)
Hypertension	33 (62.3%)	17 (51.5%)
Diabetes	35 (66.0%)	17 (51.5%)
Dyslipidemia	20 (37.7%)	19 (57.6%)
Family History	8 (15.1%)	13 (39.4%)

Male preponderance (73.6% vs. 78.8%) and smoking (58.5% vs. 39.4%) were more prevalent in the proximal LAD group, while a higher proportion of dyslipidemia (57.6% vs. 37.7%) and family history of coronary artery disease (39.4% vs. 15.1%) was associated with the mid-LAD group. These variations present varying patterns of risk factors associated with the localization of lesions, and smoking and diabetes are common among proximal LAD.

Systemic Inflammatory Biomarkers

The proximal and mid-LAD groups differed significantly with respect to the analysis of systemic inflammatory biomarkers. NLR, PLR, and CAR intermediate median values were higher with the LAD proximal group than with the LAD mid-group (3.00, 1.50, 109.75, 70.00 and 35.71, 14.00, respectively), which shows that there was a more powerful inflammation in proximal lesions.

Table 2

Systemic Inflammatory Biomarkers

LAD Segment	Parameter	Median	Minimum	Maximum
Proximal LAD	NLR	3.0000	1.75	3.78
	PLR	109.7464	70.40	202.27
	CAR	35.7111	2.62	164.17
Mid-LAD	NLR	1.5000	1.86	3.95
	PLR	70.0000	60.00	187.83
	CAR	14.0000	4.15	164.17

These raised biomarker levels in the proximal LAD group are indications of correlation with greater inflammatory processes, which seem to cause increased plaque instability and infarct size. Significant differences ($p<0.05$) between the groups in NLR and CAR, and a noteworthy difference in PLR ($p=0.08$), were confirmed by statistical analysis of the Wilcoxon signed-rank test.

Graph 1

Comparison of Median NLR, PLR, and CAR by LAD Segment

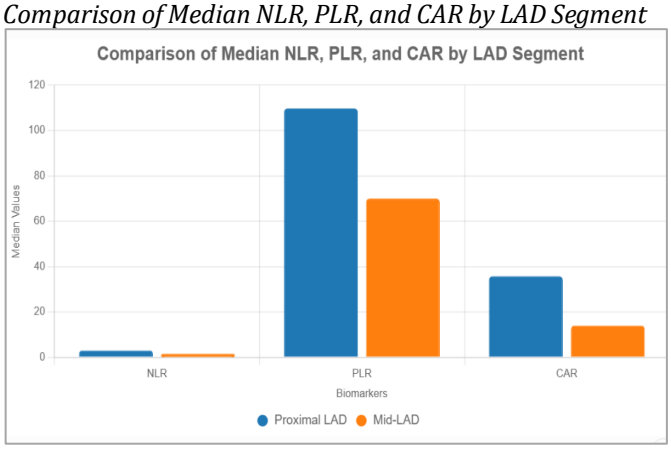

Table 3

Additional Biochemical Parameters

LAD Segment	Parameter	Median	Minimum	Maximum
Proximal LAD	WBC	20.1050	7.10	66.00
	Neutrophils	8.6000	4.10	8.70
	Lymphocytes	2.3000	2.20	2.80
	Platelets	249.0000	173.00	445.00
Mid-LAD	WBC	9.0000	8.00	51.00
	Neutrophils	5.6000	4.10	8.70
	Lymphocytes	2.2000	2.20	2.50
	Platelets	220.0000	132.00	432.00

The Spearman correlation analysis showed that NLR and CAR were positively correlated ($r=0.62$, $p<0.01$) in the proximal LAD group, indicating that these two biomarkers could be two sides of the same coin, as they could have shared the same inflammatory burden of more severe lesions. The obtained results allow emphasizing the possibility of using NLR, PLR, and CAR as the semicolon predictors of localization and severity of LAD lesions, which are proximal lesions associated with increased inflammatory activity.

DISCUSSION

These findings by the retrospective study in Dr. Ruth K.M. Pfau Civil Hospital, Karachi, indicate the possibility of a meaningful correlation between the systemic inflammatory parameters, including (NLR), (PLR) and (CAR) with the site and the severity of lesions in left anterior descending (LAD) artery in acutely presented ST-segment elevation myocardial infarction. The results of the study support a growing body of evidence that inflammation is of supreme concern with regard to pathogenesis and prognosis of acute coronary syndrome (ACS) (1). Surprisingly, the median NLR (3.00 vs. 1.50), PLR (109.75 vs. 70.00) and CAR (35.71 vs. 14.00) were remarkably higher in patients with proximal LAD lesions than in patients with MID-LAD lesions, which appears to suggest that inflammation and processes are stronger in patients with proximal lesions (2). This conclusion corresponds to other existing studies, which have revealed that higher inflammatory markers are also associated with more severity in coronary artery diseases and poor clinical outcomes (3). The differences in the prevalence of

smoking (58.5% vs. 39.4%) and diabetes (66.0% vs. 51.5%) further confirm the strong control of the interrelationship between traditional risk factors and inflammation, which may lead to further rupture of the plaque and thrombus formation (4).

The usefulness of NLR, PLR, and CAR is in the expression of systemic inflammatory reactions resulting in atherosclerotic plaque development and rupture. NLR that reflects the proportions of pro-inflammatory neutrophils and lymphocytes regulating the immune response has been continuously associated with the severity of coronary lesions and adverse cardiac events of STEMI patients (5). Greater NLR in the proximal LAD group means a more severe inflammatory condition, and in all probability, it can be caused by more intense neutrophil response and macrophage invasion of weak plaques (6). Likewise, activation of platelets, incorporated in PLR, is an indicator of increased thrombotic risk, which is a vital contributor to the evolution of ACS (7). Our proximal LAD cohort exhibited an elevated PLR that corresponds with previous findings indicating it is predictive of an increased in-hospital mortality and bigger infarct size (8). Interestingly, adding to the prognostic accuracy in cancer is a composite interventional factor, such as C-reactive protein (C-reactive protein) with a nutritional status indicator (albumin), and CAR covers both inflammatory and overall health dynamics (9). The high relationship between NLR and CAR ($r=0.62$, $p<0.01$), as observed in our study, supports their complementarity in the selection of patients with the more severe lesion of LAD (10).

The clinical importance of LAD lesion localization is explained by the fact that the artery provides a significant part of the myocardium. The LAD lesions that were proximal, like their prevalence rates in 60.9 percent of our cohort, are attributed to larger myocardial infarctions and higher chances of developing complications, including cardio-failure and irregular beating (11). These patients have higher levels of inflammatory biomarkers, which are indicative of the proximal segment and higher hemodynamic stress, or vulnerability of plaques, potential predictors of systemic inflammation dominating the progression to plaque formation or rupture (12). This observation can be proven by the works with the advanced imaging, including optical coherence tomography, which associated the amplified inflammation markers with the accumulations of macrophages and thin-cap fibroatheromas in the coronary plaques (13). The observed fewer biomarker values in the mid-LAD group can be associated with less harmful inflammatory reactions or the difference in the plaque composition, which could be causing smaller infarcts and producing more positive outcomes (14).

The implications of the study relate most to the situation in Pakistan, and in particular to coronary heart disease, which is a major cause of death in the country, and there is a lack of resources to provide advanced diagnostic tools (15). Due to cost-factor and accessibility of NLR, PLR, and CAR from a regularly taken blood test, these methods are feasible to use in low- and middle-income locations to stratify the risks (16). These biomarkers would enable immediate schemes of intervention, whether primary PCI or more aggressive medical treatment, in patients at higher risk of complications due to proximal LAD lesions in order to avoid complications such as heart failure or sudden cardiac death (17). Moreover, using such biomarkers in clinical practice might improve risk-stratification algorithms, allowing clinicians to monitor patients with the highest risk or act aggressively towards treatment (18). New biomarkers, which include uric acid-to-albumin ratio, demonstrate promise to predict outcomes in non-ST-elevation ACS, implying that an expanded array of inflammatory markers is likely to better characterize prognosis (19).

Regardless of such findings, limitations should be outlined. The retrospective study design and the comparatively small sample (N=87) can result in a lack of generalizability, especially when higher or different populations or contexts are involved. Moreover, NLR, PLR, and CAR were significantly associated with LAD lesion localization, but other confounders, such as genetic factors or unaccounted confounding variables, could impact the relationships. It may be suggested that future research be aimed towards longitudinal endpoints and the use of more advanced imaging to explain further the mechanistic connections between inflammation and coronary pathology. However, the study provides useful data to the existing scarce local evidence in Pakistan on the use of systemic inflammatory biomarkers in treating STEMI patients.

CONCLUSION

The study is a retrospective study at Dr. Ruth K.M. Pfau Civil Hospital, Karachi, which highlights the strong relationship between systemic inflammatory markers of the (NLR), (PLR), and the (CAR) and the location and extent of (LAD) artery lesions in acute (STEMI) patients The increasing NLR, PLR, and CAR were observed in those with proximal lesions of LAD on the one hand, as well as in patients with mid-LAD lesions on the other, demonstrating the presence of a more significant inflammatory process associated with a more extreme pathology of coronaries. These are convenient and low-cost biomarkers whose measurements, based on routine blood procedures, provide useful risk stratification options in resource-limited countries such as Pakistan. These markers are able to promote early interventions by targeting patients at increased risk of adverse events such as heart failure or arrhythmias. These findings need to be confirmed by further research, ideally in bigger cohorts, and examining more potential biomarkers to optimize the management of STEMI patients and thereby mitigate the burden of coronary heart disease.

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