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Original Research Article

Association Between Platelet-to-Lymphocyte Ratio and Coronary Artery Disease Severity in Chronic Coronary Syndrome

Sudipta Bakchi*[a] AKM Fazlur Rahman[b], Tanjima Parvin[b], S M Ahsan Habib[c], Faria Rahman[d], Md Noor Nabi Khandaker[e], Swadesh Kumar Saha[d], Nitay Kumar Ghosh[b], Rasedul Islam[d], Kishore kumar Shil[f]

[a] Department of Cardiology, Khulna Medical College Hospital, Khulna, Bangladesh
[b] Department of Cardiology, Bangladesh Medical University, Dhaka, Bangladesh
[c] Interventional Division of Cardiology, Bangladesh Medical University, Dhaka, Bangladesh
[d] Department of Cardiology, National Institute of Cardiovascular Disease Hospital, Dhaka, Bangladesh
[e] Department of Cardiology, Combined Military Hospital, Dhaka
[f] Department of Endocrinology, Khulna Medical College Hospital, Khulna

***Correspondence to:**

Dr. Sudipta Bakchi

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Abstract: ***Background*:** Platelet-to-lymphocyte ratio (PLR) is a novel inflammatory biomarker linked to cardiovascular outcomes. However, its association with angiographic severity of coronary artery disease (CAD) in chronic coronary syndrome (CCS) is inadequately studied in the Bangladeshi population. The study aimed to evaluate the relationship between PLR and angiographic CAD severity using the Gensini score in CCS patients. ***Methods:*** A cross-sectional analytical study was conducted in the Department of Cardiology, BSMMU, from February 2022 to January 2023. Fifty-five CCS patients undergoing elective coronary angiography were enrolled. PLR was calculated from complete blood counts, and CAD severity was assessed via Gensini scoring. Severe CAD was defined as a score ≥25. Statistical analyses included Spearman's correlation, multiple linear regression, and ROC curve analysis. ***Results:*** Severe CAD patients exhibited significantly higher PLR values (126 ± 11.8) compared to those with mild CAD (83.4 ± 16.7; $p<0.001$). PLR showed a moderate positive correlation with the Gensini score ($r=0.458$; $p<0.001$). A PLR cut-off of 106.5 predicted severe CAD with 77.8% sensitivity and 78.9% specificity (AUC 0.787). In multivariate regression analysis, PLR remained an independent predictor alongside hypertension, diabetes mellitus, LDL cholesterol, and reduced LVEF, all of which were significantly associated with severe disease burden. ***Conclusion:*** PLR is significantly associated with angiographic CAD severity and may serve as a cost-effective marker for risk stratification in CCS patients. Incorporating PLR into standard diagnostic assessments could help identify individuals at higher risk for severe disease, enabling earlier interventions and improved allocation of healthcare resources, especially in resource-limited settings.

Keywords: Platelet-To-Lymphocyte Ratio (PLR), Cardiovascular Diseases, Global Morbidity and Mortality.

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Introduction

Cardiovascular diseases (CVDs) represent the leading cause of global morbidity and mortality, responsible for approximately 17.7 million deaths each year and projected to reach 23.6 million deaths by 2030.[1, 2] Among these, coronary artery disease (CAD) constitutes the majority of cases and significantly contributes to disability-adjusted life years worldwide. CAD manifests clinically as acute coronary syndrome (ACS) and chronic coronary syndrome (CCS).[3] CCS includes a range of conditions from stable angina pectoris to post-ACS phases, highlighting the chronicity of its pathophysiological

processes.[3, 4] The development of CAD is driven by atherosclerosis, a progressive inflammatory process characterized by lipid accumulation, endothelial dysfunction, and plaque formation.[4–6] Platelets play a critical role not only in thrombosis but also in the inflammatory response that accelerates plaque progression. They release bioactive mediators such as chemokines and cytokines that amplify vascular inflammation.[7] Conversely, lymphocytes are essential modulators of adaptive immunity, and a reduction in lymphocyte count has been observed in conditions of physiological stress and systemic inflammation, correlating with adverse cardiovascular outcomes.[8, 9] The platelet-to-lymphocyte ratio (PLR) combines these two hematologic parameters, serving as a simple yet effective marker of thrombo-inflammatory status.[10] Elevated PLR has been linked to increased risk of major adverse cardiovascular events, greater lesion complexity, and higher mortality rates following percutaneous coronary interventions.[11–13] Coronary angiography remains the gold standard for diagnosing CAD and assessing disease severity. The Gensini score is a widely validated angiographic tool that quantitatively measures the extent and severity of coronary atherosclerosis by assigning weights to lesions based on their location and degree of stenosis.[14]

Multiple studies have demonstrated correlations between elevated PLR and higher Gensini scores, suggesting that PLR may serve as a surrogate marker of angiographic disease burden. However, data from South Asian populations, including Bangladesh, remain limited, despite the region's high prevalence of conventional risk factors such as diabetes, hypertension, and dyslipidemia.[15, 16] In Bangladesh, where healthcare resources are constrained, inexpensive and readily available prognostic markers are of particular value. Conventional risk assessments, though valuable, may not fully capture inflammatory contributions to CAD progression. The addition of PLR to standard evaluations could enhance early risk stratification, allowing clinicians to identify patients at higher risk for severe CAD and prioritize intensive management strategies.[17, 18] The present study aims to explore the association between PLR and angiographic CAD severity among Bangladeshi patients with CCS, using the Gensini scoring system as a validated quantitative measure of disease burden. Establishing this relationship may provide clinicians with an accessible biomarker to augment current diagnostic and prognostic frameworks, potentially improving patient outcomes in both tertiary and primary care settings.

Methods

Study Design and Participants

This observational cross-sectional study was conducted at BSMMU, Dhaka, between February 2022 and January 2023. Fifty-five CCS patients scheduled for elective coronary angiography were recruited via purposive sampling. Inclusion criteria comprised adults with CCS, while exclusions included acute infections, chronic inflammatory or hematological disorders, previous myocardial infarction or revascularization, advanced heart failure, malignancies, and pregnancy.

Data Collection

Demographic and clinical data, including age, sex, and conventional CAD risk factors (hypertension, diabetes mellitus, dyslipidemia, smoking, and family history), were documented. Laboratory parameters included complete blood count (CBC), lipid profile, serum creatinine, and echocardiographic left ventricular ejection fraction (LVEF).

Platelet-to-Lymphocyte Ratio and CAD Severity

PLR was calculated by dividing platelet count by absolute lymphocyte count from fasting venous blood samples. Coronary angiograms were interpreted by two independent cardiologists, and CAD severity was quantified using the Gensini score, with ≥25 defining severe CAD, consistent with previous studies.

Sample Size Calculation

Sample size estimation was based on detecting a significant correlation between platelet-to-lymphocyte ratio (PLR) and Gensini score among patients with chronic coronary syndrome. A two-sided alpha of 0.05 and 80% power were assumed, with an anticipated correlation coefficient of 0.37 derived from prior studies. The required sample size was calculated using the following formula:

$$n = \left(\frac{Z_\alpha + Z_\beta}{C}\right)^2 + 3$$

where $C=0.5 \times \ln\left[\frac{1+r}{1-r}\right] = 0.3884$, $Z_\alpha = 1.96$ for a 5% level of significance, $Z_\beta = 0.842$ for 80% power, and $r = 0.37$. Substitution of these values yielded an estimated sample size of approximately 47 participants. To

accommodate potential exclusions or incomplete data, this was increased by about 15%, resulting in a final required sample size of 55 participants, which was achieved in this study.

Statistical Analysis

Continuous variables were expressed as mean ± standard deviation, and categorical variables as frequencies. Group comparisons employed t-tests or chi-square tests, as appropriate. Spearman's correlation assessed PLR and Gensini score association. Multiple linear regression identified independent predictors of severe CAD. Receiver operating characteristic (ROC) analysis determined PLR cut-off for severe CAD prediction. A p-value <0.05 was considered significant. Analyses were conducted using SPSS version 23.

Results

Baseline Characteristics

In the study population, patients with severe CAD were predominantly older compared to those with mild CAD. Among individuals with mild CAD, the majority (78.9%) were aged below 50 years, whereas in the severe CAD group, most participants (52.8%) were aged 50–60 years, followed by 30.6% in the 61–70-year range. The mean age was significantly higher in the severe CAD group (60.7 ± 8.2 years) compared to the mild CAD group (48.6 ± 5.8 years, $p < 0.001$). The proportion of males was comparable between the mild and severe CAD groups (68.4% vs. 72.2%, $p = 0.768$). Smoking prevalence was similar in both groups (52.6% vs. 55.6%, $p = 0.836$). Hypertension was common in both mild (84.2%) and severe CAD (86.1%) groups ($p = 0.586$). Diabetes mellitus was more frequent in the severe CAD group (58.3%) than in the mild CAD group (31.6%), though the difference was not statistically significant ($p = 0.059$). Dyslipidemia was observed in 78.8% of the mild CAD group and 77.9% of the severe CAD group ($p = 0.920$). A family history of CAD was reported by 42.1% of participants with mild CAD and 47.2% with severe CAD ($p = 0.717$).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population

Parameter	Mild CAD (n=19)	Severe CAD (n=36)	p-value
Age Groups			<0.001
< 50 yrs.	15 (78.9%)	3 (8.3%)	
50-60 yrs.	3 (15.8%)	19 (52.8%)	
61-70 yrs.	1 (5.3%)	11 (30.6%)	
> 70 yrs.	0 (00.0%)	3 (8.3%)	
Age (years, mean ± SD)	48.6 ± 5.8	60.7 ± 8.2	<0.001
Male sex, n (%)	13 (68.4)	26 (72.2)	0.768
Smoking	10 (52.6%)	20 (55.6%)	0.836
Hypertension, n (%)	16 (84.2)	31 (86.1)	0.586
Diabetes mellitus, n (%)	6 (31.6)	21 (58.3)	0.059
Dyslipidemia	15(78.8%)	28(77.9%)	0.920
Family history of CAD	8 (42.1%)	17 (47.2%)	0.717

Laboratory Findings

In the laboratory assessment, the mean platelet count was higher in the severe CAD group (255.8 ± 34.8 × 10^3/mm^3) compared to the mild CAD group (209.9 ± 30.9 × 10^3/mm^3, $p < 0.001$). The mean WBC count was lower in the severe CAD group (7697.2 ± 890.4) than in the mild CAD group (8357.9 ± 577.7, $p = 0.005$). The mean lymphocyte count was reduced in the severe CAD group (2052.8 ± 234.8 × 10^3/mm^3) compared to the mild CAD group (2473.7 ± 579.1 × 10^3/mm^3, $p < 0.001$). The mean PLR was elevated in the severe CAD group (126.0 ± 11.8) relative to the mild CAD group (83.4 ± 16.7, $p < 0.001$). LDL cholesterol levels were slightly higher in the severe CAD group (107.7 ± 8.2 mg/dL) than in the mild CAD group (103.6 ± 5.6

mg/dL, $p = 0.045$). Serum creatinine was higher in the severe CAD group (1.1 ± 0.2 mg/dL) compared to the mild CAD group (1.0 ± 0.1 mg/dL, $p < 0.001$).

Table 2: Laboratory Findings

Parameter	Mild CAD (n=19)	Severe CAD (n=36)	p-value
Platelet count (×10³/mm³) (mean ± SD)	209.9 ± 30.9	255.8 ± 34.8	<0.001
WBC Count	8357.9 ± 577.7	7697.2 ± 890.4	**0.005**
Lymphocyte count (×10³/mm³) (mean ± SD)	2473.7 ± 579.1	2052.8 ± 234.8	<0.001
PLR (mean ± SD)	83.4 ± 16.7	126 ± 11.8	<0.001
LDL cholesterol (mg/dL) (mean ± SD)	103.6 ± 5.6	107.7 ± 8.2	0.045
Serum creatinine (mg/dL) (mean ± SD)	1.0 ± 0.1	1.1 ± 0.2	<0.001

Echocardiographic and Angiographic Findings (Table 3)

In the echocardiographic evaluation, the mean left ventricular ejection fraction (LVEF) was 61.2 ± 3.4% in the mild CAD group and 59.3 ± 6.3% in the severe CAD group, with no statistically significant difference ($p = 0.222$). The mean Gensini score was higher in the severe CAD group (44.2 ± 24.5) compared to the mild CAD group (13.05 ± 6.4, $p < 0.001$).

Table 3: Echocardiographic and Angiographic Parameters

Parameter	Mild CAD (n=19)	Severe CAD (n=36)	p-value
LVEF (%) (mean ± SD)	61.2 ± 3.4	59.3 ± 6.3	0.222
Gensini score (mean ± SD)	13.05 ± 6.4	44.2 ± 24.5	<0.001

Spearman's correlation analysis revealed a significant positive correlation between platelet-to-lymphocyte ratio (PLR) and Gensini score ($r = 0.458$; $p < 0.001$), indicating that higher PLR values were associated with higher angiographic severity scores (Figure 1).

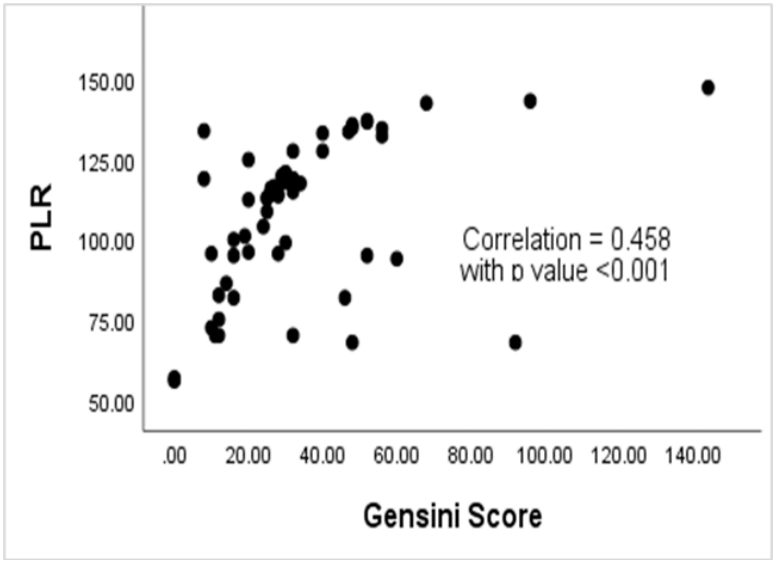

Figure 1: Correlation and Regression Analyses

Multiple Linear Regression Analysis of Determinants of Severe Coronary Artery Disease

In the multiple linear regression analysis for determinants of severe CAD (Gensini score ≥25), hypertension (B = –8.419, 95% CI: –21.283 to –4.446, $p = 0.049$), diabetes mellitus (B = –15.017, 95% CI: –29.465 to –0.569, $p = 0.042$), LDL cholesterol (B = 1.016, 95% CI: 0.029 to 2.004, $p = 0.044$), WBC count (B = –0.007,

95% CI: –0.013 to –0.001, $p = 0.023$), platelet count (B = 0.000, 95% CI: 0.000 to 0.000, $p = 0.033$), left ventricular ejection fraction (LVEF) (B = –1.717, 95% CI: –2.539 to –0.894, $p < 0.001$), and platelet-to-lymphocyte ratio (PLR) (B = 0.269, 95% CI: 0.058 to 0.480, $p = 0.014$) were statistically significant. All other variables, including age, sex, smoking, dyslipidemia, family history of CAD, total cholesterol, HDL cholesterol, triglycerides, serum creatinine, hemoglobin, and lymphocyte count, did not show statistically significant associations with severe CAD. Variance inflation factors (VIFs) for all predictors were below 5, indicating no substantial multicollinearity.

Table 4: Multiple linear regression analysis of determinants of severe CAD (Gensini score ≥25)

Variable	B	95% CI (Lower–Upper)	p-value	VIF
Age (years)	0.271	–0.394 to 0.936	0.415	2.587
Sex (male)	–5.587	–21.996 to 10.822	0.495	3.737
Smoking	–5.007	–18.556 to 8.542	0.459	3.062
Hypertension	–8.419	–21.283 to –4.446	**0.049**	1.384
Diabetes mellitus	–15.017	–29.465 to –0.569	**0.042**	3.510
Dyslipidemia	4.597	–8.349 to 17.542	0.476	1.895
Family history of CAD	5.874	–6.095 to 17.842	0.326	2.389
Total cholesterol (mg/dL)	–0.314	–0.856 to 0.227	0.247	3.142
LDL cholesterol (mg/dL)	1.016	0.029 to 2.004	**0.044**	3.730
HDL cholesterol (mg/dL)	–0.756	–3.413 to 1.900	0.568	1.790
Triglycerides (mg/dL)	0.066	–0.138 to 0.270	0.517	2.012
Serum creatinine (mg/dL)	8.523	–27.066 to 44.112	0.630	2.163
Hemoglobin (g/dL)	0.161	–8.486 to 8.807	0.970	2.900
WBC count (×10³/μL)	–0.007	–0.013 to –0.001	**0.023**	1.854
Platelet count (×10³/μL)	0.000	0.000 to 0.000	**0.033**	2.937
Lymphocyte count (×10³/μL)	–0.007	–0.019 to 0.005	0.245	1.745
LVEF (%)	–1.717	–2.539 to –0.894	**<0.001**	1.372
PLR	0.269	0.058 to 0.480	**0.014**	1.735

Note: Dependent variable: Severe CAD (Gensini score ≥25), B: Beta coefficient, significant predictors are in bold ($p < 0.05$).

ROC Analysis

Receiver operating characteristic (ROC) curve analysis established a PLR cut-off value of 106.5 for predicting severe CAD, yielding 77.8% sensitivity and 78.9% specificity (AUC 0.787; 95% CI 0.659–0.914; $p<0.001$).

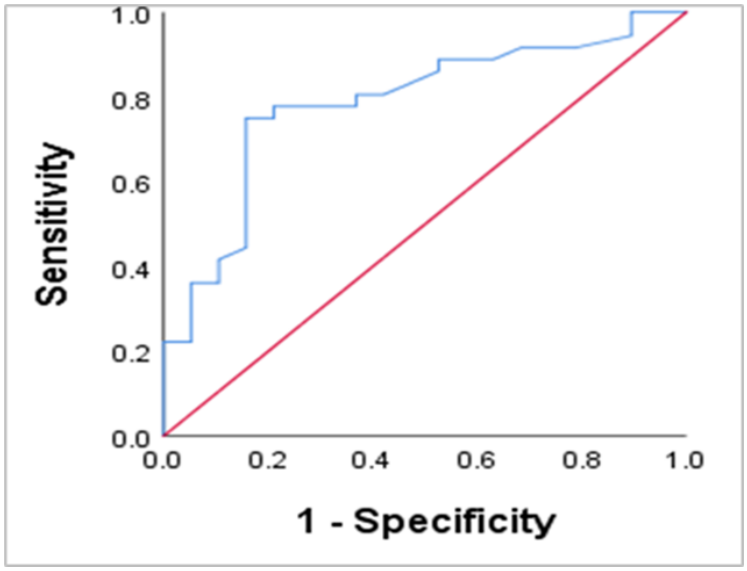

Figure 2: ROC Curve for PLR in Predicting Severe CAD

(Figure showing ROC curve with AUC 0.787, sensitivity 77.8%, specificity 78.9%.)

Discussion

The present study demonstrates a moderate but statistically significant association between PLR and angiographic severity of CAD in patients with chronic coronary syndrome (CCS). Patients with severe CAD, defined by a Gensini score ≥25, exhibited markedly elevated PLR compared to those with mild CAD. This finding supports the hypothesis that PLR serves as an integrated marker of both pro-thrombotic and pro-inflammatory processes driving atherosclerotic disease progression. Mechanistically, elevated platelet counts contribute to atherogenesis through release of inflammatory mediators, adhesion molecules, and pro-thrombotic factors that facilitate plaque growth and instability. Conversely, reduced lymphocyte counts reflect heightened physiological stress and immune dysregulation, both of which have been associated with adverse cardiovascular outcomes. The PLR thus unifies these opposing hematological trends into a single parameter that mirrors systemic vascular inflammation and thrombogenic potential, aligns with previously reported findings.[19] Our findings align with earlier studies by Yuksel *et al*. and Akboga *et al*., which similarly reported that elevated PLR correlates with increased angiographic burden of CAD and independently predicts adverse cardiac events.[20, 21] The PLR threshold identified in our study (106.5) closely approximates values reported in international literature, though slight variation likely reflects ethnic differences, baseline risk factor profiles, and methodological variations in CAD severity assessment.

In the Bangladeshi context, where resource constraints limit access to advanced inflammatory biomarkers such as high-sensitivity C-reactive protein (hs-CRP), PLR offers a readily available and cost-effective alternative derived from routine complete blood count testing. Given the high prevalence of diabetes, hypertension, and dyslipidemia in this population, integrating PLR into risk stratification frameworks could enable earlier identification of patients with severe disease who may benefit from intensified medical therapy or revascularization.[22–24] Conventional risk factors in this study, including diabetes and hypertension, were more prevalent among severe CAD patients, consistent with global epidemiological patterns. However, PLR remained an independent predictor even after adjusting for these confounders, underscoring its incremental prognostic utility beyond established risk factors. The positive correlation with Gensini score further supports its role as a surrogate marker of total atherosclerotic burden rather than focal stenosis alone. [25, 26]

In our multivariable analysis, hypertension, diabetes mellitus, LDL cholesterol, WBC count, platelet count, and reduced LVEF also emerged as significant predictors of severe CAD, consistent with established

evidence on their role in atherosclerotic progression and cardiovascular outcomes.[27,28] Despite these strengths, certain limitations merit consideration. The study's single-center design and modest sample size may restrict generalizability. Its cross-sectional nature precludes causal inference, and dynamic changes in PLR overtime were not assessed. Additionally, comparative evaluation against other inflammatory ratios, such as neutrophil-to-lymphocyte ratio (NLR), was not performed. Future multicenter prospective studies are warranted to validate these findings and explore PLR's integration into composite risk scores and predictive algorithms for CAD severity and outcomes. In summary, this study provides compelling evidence supporting PLR as a clinically useful, inexpensive biomarker for assessing CAD severity in CCS patients. Its application may be particularly impactful in low-resource settings, aiding clinicians in refining diagnosis, risk stratification, and therapeutic decision-making.

Conclusion

PLR is independently associated with angiographic CAD severity in CCS and offers a cost-effective, readily available biomarker for risk stratification. Incorporation of PLR into clinical assessment may facilitate early identification of high-risk patients and guide management strategies in resource-limited settings.

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