Journal of Health and Nutrition Research
Vol. 5, No. 1, 2026, pg. 443-449, https://doi.org/10.56303/jhnresearch.v5i1.951
Journal homepage: https://journalmpci.com/index.php/jhnr/index

e-ISSN: **2829-9760**

Correlation Between Platelet-to-Lymphocyte Ratio and Low-Density Lipoprotein Cholesterol Levels in Patients With Coronary Heart Disease

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ORIGINAL ARTICLES

Submitted: 15 October 2025

Accepted: 23 February 2026

Keywords:
Coronary Heart Disease, Dyslipidemia, Inflammation, Low-Density Lipoprotein Cholesterol, Platelet-to-Lymphocyte Ratio

ABSTRACT

Coronary heart disease (CHD) remains a leading cause of mortality worldwide and is primarily driven by atherosclerosis, a pathological process in which dyslipidemia and chronic inflammation play central roles. Elevated low-density lipoprotein (LDL) cholesterol is a well-established risk factor for atherogenesis, contributing to lipid accumulation within the arterial wall and subsequent plaque formation. In parallel, inflammatory mechanisms are critically involved in plaque progression and instability, underscoring the importance of inflammatory biomarkers in cardiovascular research. The platelet-to-lymphocyte ratio (PLR) has emerged as a simple and cost-effective hematological marker that reflects systemic inflammation and prothrombotic activity. Several studies have reported an association between elevated PLR and adverse cardiovascular outcomes. However, the relationship between PLR and lipid parameters, particularly LDL cholesterol, remains unclear. Previous findings have been inconsistent, and limited evidence is available regarding this association in patients with stable and medically treated CHD. Given these uncertainties, further investigation is warranted to clarify the relationship between inflammatory markers and lipid profiles in stable CHD populations. Therefore, this study aimed to evaluate the correlation between the platelet-to-lymphocyte ratio and LDL cholesterol levels in patients with stable coronary heart disease.

Key Messages:

- LDL cholesterol was significantly higher in patients with stable CHD, confirming dyslipidemia as a key factor in the disease.
- No significant correlation was found between PLR and LDL levels in this cohort of stable CHD patients.
- In stable and medically treated CHD patients, PLR may not be a reliable marker of lipid-associated inflammation.
- Standard treatments for CHD may mask the underlying relationship between inflammatory markers and lipid levels.

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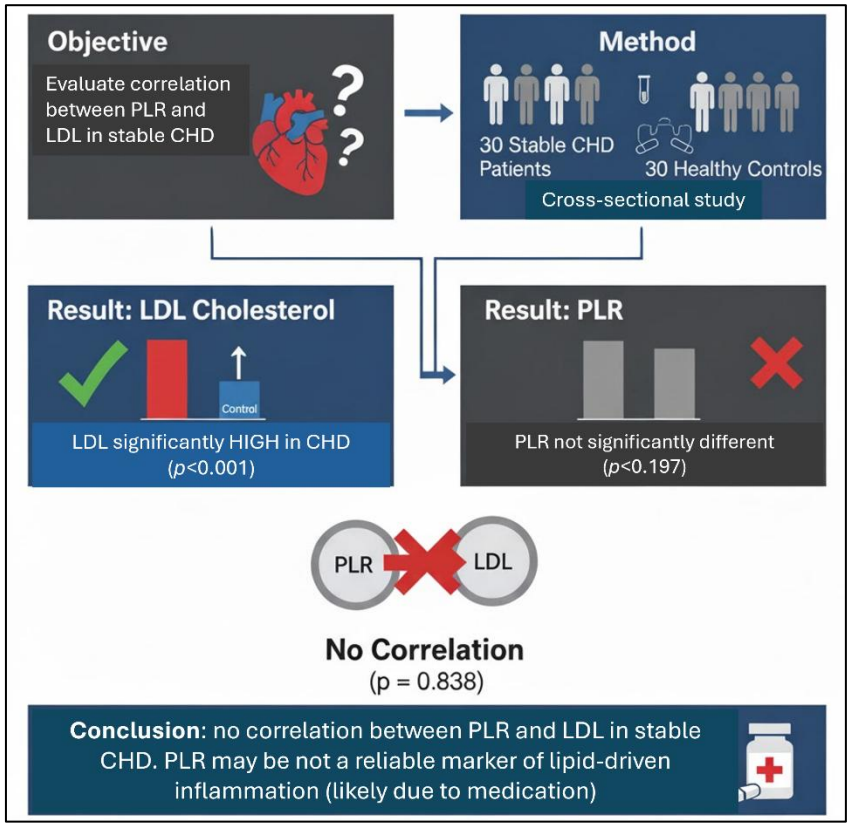
GRAPHICAL ABSTRACT



INTRODUCTION

Coronary heart disease (CHD) is the leading cause of death globally, responsible for approximately 17.9 million deaths in 2019 (1). The disease arises from the narrowing of coronary arteries, which reduces blood supply to the heart muscle (2). This process is primarily driven by atherosclerosis, a condition characterized by the dual pathologies of chronic inflammation and lipid accumulation within the arterial wall (3). With the incidence of CHD rising in Indonesia, there is an urgent need for better risk assessment strategies.

Two key components of atherosclerosis can be measured using accessible biomarkers. The first, dyslipidemia, is a major risk factor characterized by elevated low-density lipoprotein (LDL) cholesterol (4). LDL promotes lipid deposition in arteries, leading to endothelial dysfunction and the formation of atherosclerotic plaques. The rupture of these plaques can cause thrombus formation and trigger acute coronary events (5). The second component, inflammation, can be assessed using simple hematological indices like the platelet-to-lymphocyte ratio (PLR). The PLR is a cost-effective marker that reflects both systemic inflammation and a pro-thrombotic state, and elevated PLR has been linked to adverse cardiovascular outcomes (6). However, the relationship between the inflammatory marker PLR and the lipid marker LDL remains unclear, with previous studies showing inconsistent results (7–9). Some research finds a positive correlation, suggesting PLR reflects lipid-driven inflammation, while others report no significant link. These discrepancies may be due to differences in patient populations, disease stage, or medication use (10,11).

This study aimed to determine the relationship between the platelet-to-lymphocyte ratio and LDL cholesterol levels in patients with coronary heart disease. The research included 60 participants, consisting of 30 patients diagnosed with CHD and 30 healthy individuals as controls. The findings are expected to provide insight into the relationship between inflammation and lipid metabolism in CHD and support the potential use of PLR as an additional marker in cardiovascular risk assessment.

https://doi.org/10.56303/jhnresearch.v5i1.951

METHODS

Study Design

This study employed a retrospective design with a cross-sectional approach. The aim was to determine the correlation between the platelet-to-lymphocyte ratio (PLR) and low-density lipoprotein (LDL) cholesterol levels in patients diagnosed with coronary heart disease (CHD). This study employed a retrospective design with a cross-sectional approach. The aim was to determine the correlation between the platelet-to-lymphocyte ratio (PLR) and low-density lipoprotein (LDL) cholesterol levels in patients diagnosed with coronary heart disease (CHD). This research received permission from Mitra Keluarga Hospital Surabaya, as stated in the letter number 221/SBY-DIR/EKS/VII/2024.

Samples, Inclusion and Exclusion Criteria

This study enrolled 60 CHD outpatients at Mitra Keluarga Surabaya Hospital from September to November 2023. These patients were divided into 2 groups, 30 patients with CHD and 30 healthy negative control groups. The inclusion criteria are patients diagnosed with coronary heart disease and dyslipidemia, underwent complete blood count and LDL cholesterol testing, aged 45–59 years, who were obese or overweight, fasting 8–12 hours prior to blood sampling, receiving regular treatment for at least one year. The exclusion criteria are patients who had undergone LDL cholesterol testing after receiving lipid-lowering therapy, patients with incomplete or missing clinical or laboratory data, patients with uncontrolled lifestyle factors, and active or passive smokers.

Data Collection Procedures

Venous blood samples were collected using standard phlebotomy techniques. Approximately 3–5 mL of blood was drawn using a vacutainer system. Blood was collected into EDTA tubes for hematological analysis and serum-separating tubes (SST) for biochemical analysis. Serum was obtained after centrifugation at 3000 rpm for 20 minutes.

Instruments and Materials

Sysmex XN-1000 (Sysmex Corporation, Japan) was used to determine platelet and lymphocyte counts through automated hematology analysis. Thermo Fisher Indiko Plus (Thermo Fisher Scientific, Finland) was used to measure LDL cholesterol using enzymatic colorimetric methods. All reagents and calibrators were verified according to manufacturer protocols, and daily quality control was performed using standardized QC materials.

Data Processing and Analysis

Data on platelet counts, lymphocyte counts, and LDL cholesterol levels were tabulated from the blood samples of outpatients with CHD in Mitra Keluarga Hospital Surabaya. The PLR was calculated by dividing platelet count ($\times 10^3/\mu L$) by lymphocyte count ($\times 10^3/\mu L$). Data normality was tested using the Shapiro–Wilk test. Statistical analysis was performed using SPSS version 25.0 (IBM Corp., USA). A p-value < 0.05 was considered statistically significant.

RESULTS

A total of 60 participants were included in this study, comprising 30 patients with coronary heart disease (CHD) and 30 healthy controls. The majority of patients in the CHD group were male (83.3%) and aged between 45–59 years. Descriptive statistics for the platelet-to-lymphocyte ratio (PLR) and low-density lipoprotein (LDL) cholesterol for both groups are presented in Table 1. The mean LDL cholesterol level was markedly higher in CHD patients (139.12±24.19 mg/dL) compared to the healthy control group (87.98±8.99 mg/dL). For context, normal LDL concentrations are below 100 mg/dL. In contrast, the mean PLR was similar between the CHD group (144.81±50.49) and the control group (157.01±51.74).

https://doi.org/10.56303/jhnresearch.v5i1.951

Table 1. Descriptive Statistics of Participant Laboratory Data

Variable	Group	Mean	SD	Min	Max
PLR	CHD Patients	144.81	50.5	48.1	323.2
PLR	Control Group	157.01	51.7	82.6	316.2
LDL (mg/dL)	CHD Patients	139.12	24.2	110	261
LDL (mg/dL)	Control Group	87.98	8.99	60	99

PLR: platelet-to-lymphocyte ratio; LDL: low-density lipoprotein; CHD: coronary heart disease

A summary of the statistical analyses is shown in Table 2. The Shapiro-Wilk test indicated that the data for both PLR and LDL were not normally distributed in either group (p<0.05). Therefore, non-parametric tests were used for subsequent analyses. A Mann-Whitney U test confirmed a statistically significant difference in LDL cholesterol levels between the CHD patients and the control group (p<0.001). The same test, however, showed no significant difference in the PLR between the two groups (p=0.197). Furthermore, a Spearman correlation test was performed to assess the relationship between the two variables within the CHD group. The results revealed no significant correlation between the PLR and LDL cholesterol levels (p=0.838, r=0.023), suggesting that in this cohort, higher LDL levels were not associated with a corresponding increase in PLR.

Table 2: Summary of Statistical Analyses

Analysis Type	Variables	Significance (p-value)	Interpretation
Normality Test (Shapiro-Wilk)	PLR (CHD)	0.003	Data is not normally distributed.
	PLR (Control)	0.002	Data is not normally distributed.
	LDL (CHD)	<0.001	Data is not normally distributed.
	LDL (Control)	<0.001	Data is not normally distributed.
Group Comparison (Mann-Whitney U)	PLR (CHD vs. Control)	0.197	No significant difference between groups.
	LDL (CHD vs. Control)	<0.001	Significant difference between groups.
Correlation Test (Spearman)	PLR vs. LDL (in CHD)	0.838	No significant correlation.

PLR: platelet-to-lymphocyte ratio; LDL: low-density lipoprotein; CHD: coronary heart disease

DISCUSSION

This study investigated the relationship between the platelet-to-lymphocyte ratio (PLR) and LDL cholesterol in patients with stable coronary heart disease (CHD). The key findings were that while LDL cholesterol was significantly elevated in CHD patients compared to controls, their PLR values were not. Ultimately, no significant correlation was found between PLR and LDL levels in this patient group.

The demographic profile of the CHD cohort aligns with established risk factors for the disease. The majority of patients were aged 45–59, supporting age as a major non-modifiable risk factor (12). The natural aging process can lead to endothelial dysfunction and the progressive buildup of atherosclerotic plaques (13). Furthermore, male patients predominated in the CHD group (83.3%), a finding consistent with literature suggesting men have a significantly higher risk of developing CHD (14). This elevated risk is often attributed to a combination of hormonal factors and lifestyle habits, such as smoking, which accelerates atherosclerosis (15,16).

The results showed a clear divergence between the lipid and inflammatory markers. As expected, LDL cholesterol levels were significantly higher in CHD patients than in healthy controls. This observation reinforces the critical role of dyslipidemia as a primary driver of atherosclerosis, where excess LDL contributes to plaque formation in arterial walls (17). Conversely, there was no significant difference in PLR between the CHD patients and the control group. This may be because the study participants were in a stable clinical condition (18). Other research indicates that an elevated PLR is more strongly associated with acute or unstable coronary events, where inflammation and platelet activation are markedly higher, rather than with stable CHD (19,20).

https://doi.org/10.56303/jhnresearch.v5i1.951

The study's central finding was the absence of a significant correlation between PLR and LDL cholesterol levels. A significant increase in LDL was not met with a proportional increase in PLR. While this may suggest that lipid-driven inflammation was not reflected by PLR in this stable cohort, a more likely explanation is the confounding effect of medication, as most participants were presumably receiving treatment with both aspirin and atorvastatin (21).

Although LDL cholesterol levels were significantly elevated in patients with stable coronary heart disease, this increase was not accompanied by a corresponding rise in the platelet-to-lymphocyte ratio (PLR), resulting in no significant correlation between the two variables. This finding indicates that lipid-associated inflammatory activity in stable CHD may be influenced by factors beyond isolated hematological indices. Evidence from the Journal of Health and Nutrition Research suggests that dyslipidemia and inflammation are strongly modulated by lifestyle and metabolic determinants, including poor dietary patterns, physical inactivity, and obesity, which are closely linked to metabolic syndrome and chronic low-grade inflammation (22). In addition, cardiometabolic risk factors such as elevated body mass index, hypertension, and diabetes have been shown to substantially increase coronary heart disease risk and to influence lipid metabolism and systemic inflammatory pathways (23). Interventions targeting nutrition, physical activity, and mental health have also been reported to improve cardiovascular outcomes by favorably modulating lipid profiles and inflammatory status (24). Taken together, these findings support the present results and suggest that, in stable and medically treated CHD patients, PLR has limited sensitivity as a marker of lipid-associated inflammation.

This study has several limitations. The relatively small sample size and the inclusion of only stable CHD patients are limitations that the generalizability of the findings. Data from patients with acute coronary syndromes might have revealed a stronger correlation. Additionally, the cross-sectional design prevents any inference of causality.

This study has several limitations that should be considered when interpreting the findings. The relatively small sample size may reduce statistical power and limit the generalizability of the results. In addition, the cross-sectional design precludes causal inference between LDL cholesterol levels and PLR. However, several measures were implemented to minimize potential sources of bias. The study population was restricted to patients with stable coronary heart disease who met strict inclusion and exclusion criteria, thereby reducing clinical heterogeneity. Laboratory measurements were performed using standardized automated analyzers with routine quality control procedures, minimizing measurement bias. Furthermore, non-parametric statistical methods appropriate for non-normally distributed data were applied to reduce analytical bias. Although residual confounding particularly related to medication use and lifestyle factors cannot be entirely excluded, the homogeneity of the study population and consistent treatment status were intended to mitigate these effects.

Future studies with larger sample sizes and longitudinal designs are warranted to confirm these findings and better establish causal relationships. Such studies should track the temporal relationship between lipid and inflammatory markers and include a broader spectrum of coronary heart disease severity, as well as additional inflammatory biomarkers, such as C-reactive protein (CRP) and interleukins, to provide a more comprehensive understanding of the complex interplay between inflammation and lipid metabolism in CHD.

CONCLUSION

In patients with stable coronary heart disease (CHD), this study found no significant correlation between the platelet-to-lymphocyte ratio (PLR) and low-density lipoprotein (LDL) cholesterol, despite significantly higher LDL levels in the CHD group. This suggests PLR is not a reliable marker of lipid-associated inflammation in stable, treated CHD, likely due to the confounding effects of medication. Future longitudinal studies using larger samples and additional biomarkers are recommended to clarify this relationship.

FUNDING

This research received no external funding.

https://doi.org/10.56303/jhnresearch.v5i1.951

ACKNOWLEDGMENTS

The author would like to express sincere gratitude to Universitas Maarif Hasyim Latif, Sidoarjo, and the Mitra Keluarga Hospital Surabaya for providing administrative and technical support during data collection and laboratory analysis. Appreciation is also extended to the academic supervisors and laboratory staff for their valuable guidance and assistance throughout this research project.

CONFLICTS OF INTEREST

The author declares no conflict of interest.

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