Original Article

Analysis of Platelet Large Cell Ratio (P-LCR) and its Correlation with Lipid Parameters in a Dyslipidemic Patient: A Retrospective Observational Study

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Abstract

Background: Platelet activation is among the key variables in atherothrombosis. The percentage of large, hyperreactive platelets can be measured using the Platelet Large Cell Ratio (P-LCR), a metric reported by automated hematology analyzers. More study is essential to determine the relationship with dyslipidemia, a major cardiovascular risk factor. For the hematological workup of the patients, we have the Erba Elite 580, a fully automated 5-part differential hematology analyzer designed for high-throughput complete blood count (CBC) and differential testing. The analyzer reports 26 key parameters, including: WBC, LYM (absolute and %), MON (absolute and %), NEU (absolute and %), EOS (absolute and %), BAS (absolute and %), RBC, HGB, hematocrit (HCT), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), red cell distribution width (RDW-CV and RDW-SD), PLT, platelet distribution width (PDW), mean platelet volume (MPV), plateletcrit (PCT), platelet-large cell ratio (P-LCR), and platelet-large cell count (P-LCC). Additional features include automatic floating discriminators for detecting immature or atypical cells and enhanced platelet parameters (e.g., PDW-SD, PDW-CV) for improved diagnostic accuracy. The goal of this research was to review the relationship between P-LCR and other platelet indices and standard lipid parameters by examining results in a diverse patient cohort. **Material and Methods:** One hundred patients were included in our retrospective analysis. This data included platelet indices (Platelet Count, MPV, PDW, P LCR, P-LCC) and lipid profiles (Total Cholesterol, TG, HDL, LDL, VLDL). This statistical analysis included descriptive statistics, group comparisons (t-tests and ANOVAs), and Pearson correlations. For the hematological workup of the patients, we have been using the hematological analysis Erba Elite 580. It is a fully automated 5-part differential hematology analyzer designed for high-throughput complete blood count (CBC) and differential testing. A cyanide-free reagent for hemoglobin (HGB) measurement and the impedance method for counting and sizing white blood cells (WBC), red blood cells (RBC), and platelets (PLT) have been used to guarantee operator safety and environmental compliance. It integrates chemical dye staining, flow cytometry (FCM) technology, a semiconductor laser scatter, and an independent basophil channel for WBC differential analysis. This allows for accurate 5-part differentiation (lymphocytes [LYM], monocytes [MON], neutrophils [NEU], eosinophils [EOS], and basophils [BAS]) and sophisticated 3D scatterplot visualization for morphological evaluation. The cohort's mean P-LCR was 41.79% (SD: 10.31). MPV ($r=0.994$, $p<0.0001$) and PDW-SD ($r=0.926$, $p<0.0001$) showed a significant, strong positive correlation with P-LCR. P-LCR showed a weak but significant positive correlation with HDL ($r=0.261$, $p=0.009$) and total cholesterol ($r=0.199$, $p=0.047$). P-LCR did not differ significantly between sexes ($p=0.299$) or between age groups ($p=0.200$). Platelet count was strongly negatively correlated with P-LCR ($r=-0.518$, $p<0.0001$). **Conclusion:** Elevated P-LCR is prevalent in this patient cohort and is significantly correlated with key lipid parameters. The inverse relationship between platelet count and MPV, and the strong association between MPV and platelet reactivity, suggest a state of increased platelet reactivity. P-LCR is a valuable, readily available hematological marker that may aid in assessing thrombotic risk in patients with dyslipidemia.

Keywords: Platelet Larger Cell Ratio (P-LCR), Mean Platelet Volume (MPV), Dyslipidemia, Platelet Indices, Cardiovascular Risk, Platelet Reactivity.

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Introduction

The most common cause of death and disease worldwide is still cardiovascular disease and its thrombotic complications. An established, controllable risk factor for both damaged endothelial cells and the development of plaque is dyslipidemia, which is characterized by high levels of triglycerides, LDL, and total cholesterol, or by the absence

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of HDL.[1] Platelets actively promote atherothrombosis rather than merely serving as spectators. Vascular disease worsens when activated platelets attach to damaged endothelium, releasing and secreting pro-inflammatory and pro-thrombotic mediators.[2]

One accepted surrogate measure of platelet activity is platelet size, which is known as Mean Platelet Volume (MPV). Larger platelets have denser granules, produce more prothrombotic substances, such as thromboxane A2, and are biologically and metabolically more active.[3] Elevated MPV is associated in numerous studies with a higher risk of myocardial infarction, stroke, and ischemic heart disease.[4] However, pre-analytical factors, such as the type of anticoagulant used and the time interval between data collection and analysis, can affect MPV measurement.[5]

A further measurement offered by modern automated hematology tests is the Platelet Large Cell Ratio (P-LCR). It directly measures the most active platelet subpopulation by counting platelets that exceed a given volume threshold, typically 12 fL. Although studies such as Grotto & Noronha's have found elevated P-LCR among people with dyslipidemia,[6] additional studies are needed to determine how it relates to a full lipid profile and other social factors in a broader clinical setting. The P-LCR values and other platelet indices in a cohort of 100 patients are analyzed in this retrospective study to clarify how they relate to age, sex, and lipid parameters.

Materials and Methods

Study Design and Population: A retrospective, observational study was conducted by reviewing the laboratory records of 100 patients. The cohort comprised 64 females and 36 males, with ages varying from 1 to 73. For analytical purposes, our patients were stratified into four different age groups: <20 years (n=8), 20-40 years (n=43), 40-60 years (n=34), and 60-80 years (n=15).

Laboratory Measurements

1. The Erba ELite 580 is a fully automated five-part differential hematology analyzer, which is intended for high-throughput complete blood count (CBC) and differential testing, and it was used for the hematological analyses. To ensure operator safety and environmental compliance, this device measures hemoglobin (HGB) with a cyanide-free reagent while counting and sizing white blood cells (WBC), red blood cells (RBC), and platelets (PLT) using impedance. It combines flow cytometry (FCM) technology with a semiconductor laser scatter, chemical dye staining, and an independent basophil channel for WBC differential analysis. This allows for accurate 5-part differentiation (lymphocytes [LYM], monocytes [MON], neutrophils [NEU], eosinophils [EOS], and basophils [BAS]) and sophisticated 3D scatterplot visualization for morphological evaluation.
2. Dipotassium Ethylenediaminetetraacetic acid (K2-EDTA) vacuum tubes were used to collect venous blood samples. Calculated LDL and VLDL cholesterol, total cholesterol (CHOL), triglycerides (TG), and HDL cholesterol (HDL) were included in the lipid profile that was measured. An automated hematology analyzer was used to perform a complete blood count with platelet indices along with Platelet Count (P. COUNT), Mean Platelet Volume (MPV), Platelet Distribution Width (Standard Deviation - PWD-SD and Coefficient of Variation - PWD-CV), Platelet Larger Cell Ratio (P-LCR), and Platelet Larger Cell Count (P-LCC). Every procedure follows standard operating procedures.

Statistical Analysis: The correct statistical software was employed to analyze the data. The mean ± standard deviation was employed to express continuous variables. To evaluate differences between two groups (sex), a t-test for independent samples was used. To assess variations across many groups (age), a one-way Analysis of Variance (ANOVA) was performed. The strength and direction of linear correlations between variables were determined by applying Pearson's correlation coefficient (r). A p-value of below 0.05 is considered statistically significant.

Results

Descriptive Statistics:

The mean values and standard deviations for the entire cohort's lipid and platelet parameters are summarized in [Table 1]. The mean P-LCR was 41.79% ± 10.31.

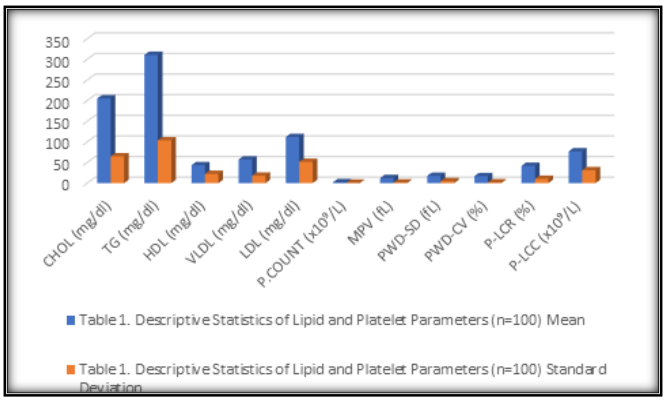
Table 1: Descriptive Statistics of Lipid and Platelet Parameters (n=100)

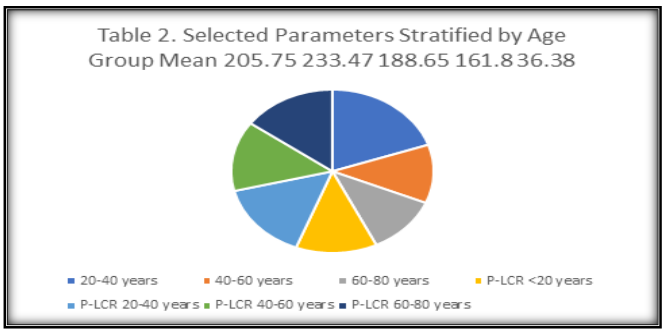
Parameter	Mean	Standard Deviation
CHOL (mg/dl)	205.26	64.92
TG (mg/dl)	310.72	103.70
HDL (mg/dl)	43.54	22.32
VLDL (mg/dl)	57.26	17.87
LDL (mg/dl)	111.76	51.65
P.COUNT (x10⁹/L)	2.14	0.93
MPV (fL)	12.07	1.48
PWD-SD (fL)	17.60	4.59
PWD-CV (%)	16.74	1.56
P-LCR (%)	41.79	10.31
P-LCC (x10⁹/L)	76.80	31.23

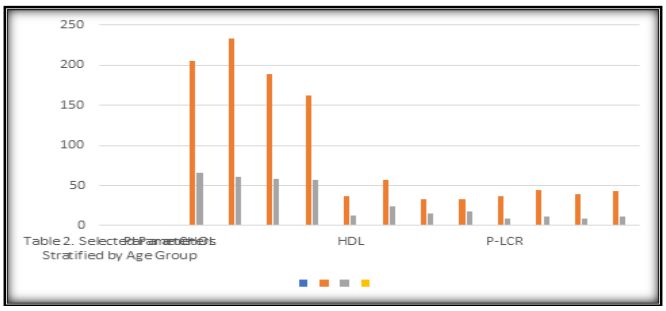
Analysis by Age and Sex: When stratified by age, the 20-40 years age group had the highest mean P-LCR (43.78%), though the differences across groups were not statistically significant (p=0.200). Significant differences were observed across age groups for CHOL, HDL, LDL, and PWD-CV (p<0.05 for all). The results are shown in detail in [Table 2].

Table 2: Selected Parameters Stratified by Age Group

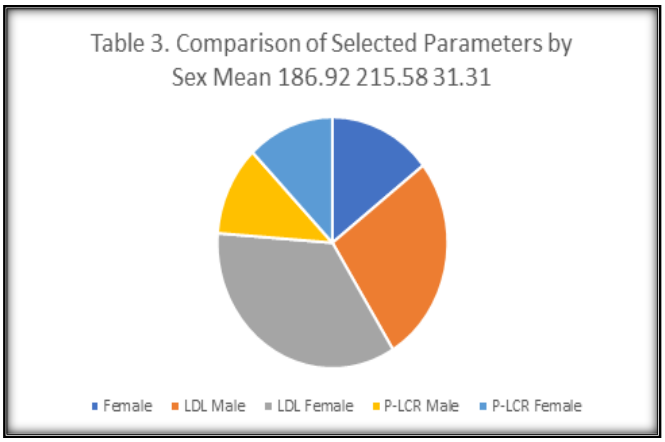
Parameter	Age Group	Mean	SD	p-value
CHOL	<20 years	205.75	66.27	0.0004
	20-40 years	233.47	60.60	
	40-60 years	188.65	58.80	
	60-80 years	161.80	57.50	
HDL	<20 years	36.38	13.20	<0.0001
	20-40 years	56.63	23.58	
	40-60 years	33.26	14.95	
	60-80 years	33.13	17.65	
P-LCR	<20 years	37.24	8.57	0.200
	20-40 years	43.78	10.99	
	40-60 years	39.79	9.09	
	60-80 years	43.04	11.09	







When males and females were compared, females had significantly higher mean levels of LDL (p=0.007), HDL (p<0.0001), and CHOL (p=0.033). P-LCR, however, did not differ statistically significantly between males (40.36%) and females (42.60%) (p=0.299). Table 3 displays the findings of the t-test analysis.



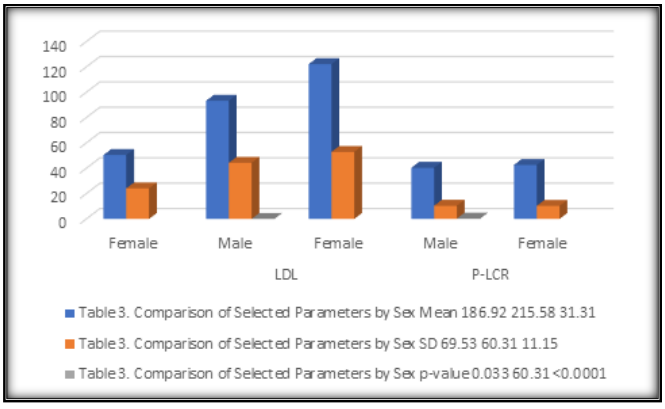


Table 3: Comparison of Selected Parameters by Sex

Parameter	Sex	Mean	SD	p-value
CHOL	Male	186.92	69.53	0.033
	Female	215.58	60.31	
HDL	Male	31.31	11.15	<0.0001
	Female	50.42	24.08	
LDL	Male	93.26	44.31	0.007
	Female	122.17	52.88	
P-LCR	Male	40.36	10.39	0.299
	Female	42.60	10.26	

Correlation Analysis

Several important conclusions were made in the correlation matrix (Table 4):

MPV (r=0.994, p<0.0001) and PWD-SD (r=0.926, p<0.0001) showed a significantly and strongly positive correlation with P-LCR.

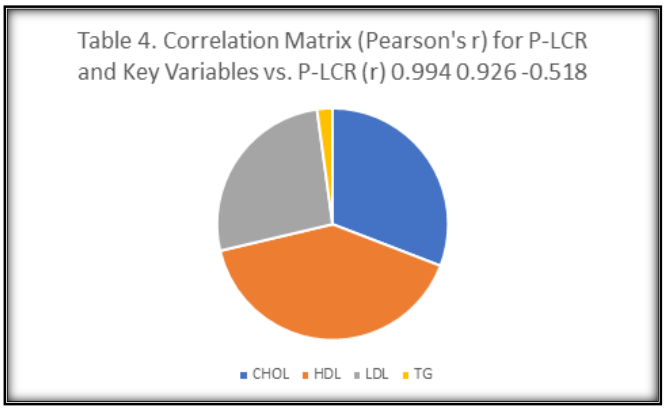
Platelet count and P-LCR have a significant negative correlation (r = -0.518, p < 0.0001).

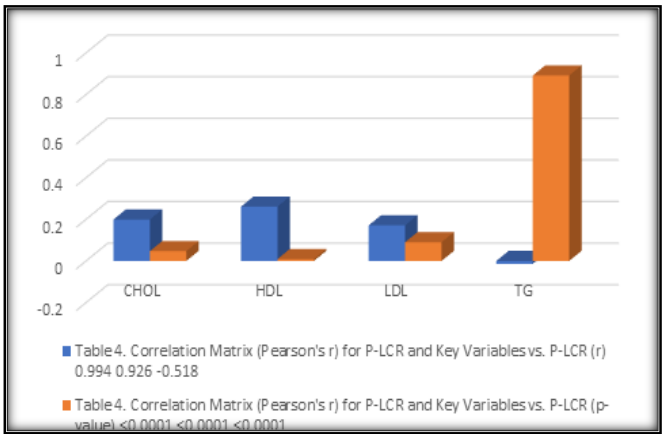
Regarding lipid parameters, P-LCR showed a weak but significant positive correlation with HDL (r=0.261, p=0.009) and CHOL (r=0.199, p=0.047).

Similar correlations have been found between MPV and HDL (r=0.266, p=0.008) and between MPV and CHOL (r=0.197, p=0.049).

Table 4: Correlation Matrix (Pearson's r) for P-LCR and Key Variables

Variable	vs. P-LCR (r)	vs. P-LCR (p-value)
MPV	0.994	<0.0001
PWD-SD	0.926	<0.0001
P.COUNT	-0.518	<0.0001
CHOL	0.199	0.047
HDL	0.261	0.009
LDL	0.171	0.090
TG	-0.014	0.891





Discussion

This is a 100-patient retrospective study that offers a thorough examination of the connection between dyslipidemia and platelet indices, specifically P-LCR. Our results support and build upon earlier studies, including the groundbreaking study by Grotto & Noronha,[6] which initially identified elevated P-LCR in dyslipidemic patients.

Our cohort's mean P-LCR was 41.79%, which was significantly higher than the previous study's control group (23.2%).[6] This indicates a widespread presence of larger, possibly more reactive platelets in our study patient population. The most important discovery was the extraordinarily high positive correlation (r = 0.994) between P-LCR and MPV. The significant negative correlation between P-LCR and platelet count (r=-0.518) is a well-documented phenomenon in hematology. It suggests a potential compensatory mechanism in thrombopoiesis, in which the bone marrow may release fewer but larger and more reactive platelets in response to various stimuli, including increased consumption or inflammatory states common in dyslipidemia.[7]

Notably, small but scientifically significant positive correlations were observed between P-LCR and HDL and total cholesterol. The relationship between total cholesterol and the prothrombotic state that hypercholesterolemia generates is well established. The relationship with cholesterol levels is more complex. Although HDL is widely accepted as cardioprotective, its link to platelet function remains unclear. The qualitative performance of HDL can be affected in a dyslipidemic state, or this finding may indicate a specific feature of our cohort that warrants further research.

The absence of obvious differences in P-LCR across age groups and sexes indicates that elevated P-LCR is strongly associated with the underlying metabolic state of dyslipidemia rather than with social variables. This increases its utility as a general-purpose biomarker to measure platelet-related risk in this patient group. The retrospective, single-center design of this study is a limitation. You fail to establish causality or set precise risk thresholds due to the lack of a healthy control population and clinical data on outcomes (such as the incidence of thrombotic events).

Conclusion

Elevated P-LCR was common in this group and showed significant correlations with lipid parameters and other recognized markers of platelet activation, such as MPV. Its function as a marker of altered platelet production and reactivity is further supported by its inverse relationship with platelet count. P-LCR offers useful, practical insight into subjective platelet abnormalities and is a parameter that is easy to measure and commonly reported by modern hematology analyzers. To more accurately stratify cardiovascular and coagulation risk in patients with dyslipidemia, we advise using P-LCR in routine screening.

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Conflicts of interest
There are no conflicts of interest.

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