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# Prediction of coronary artery disease severity from platelet to lymphocyte ratio

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**Abstract**
**Background:** Previous reviews confirmed the fundamental relation between numerous hematologic variables particularly neutrophil/ lymphocyte ratio and coronary artery disease (CAD). Predictive and more notable prognostic significance of the neutrophil/lymphocyte ratio has been clearly shown in several cardiac and vascular diseases. The goals of this research include determining the platelet to lymphocyte ratio's (PLR) values as a measure of the extent of coronary atherosclerosis, determining the correlation between PLR and atherosclerosis severity in CAD, and determining the PLR cut-off value that indicates severe CAD.
**Methods:** This case-controlled work was performed on 180 individuals planned for elective diagnostic coronary angiography. The participants were split into three equal groups regarding coronary angiography and Gensini score. Group I: 60 participants with normal coronary angiography and zero Gensini score. Group II: 60 participants with mild atherosclerotic CAD and Gensini scores of <25 points. Group III: 60 participants with severe atherosclerotic CAD and Gensini scores of ≥ 25 to detect the value of PLR as a predictor of severity of atherosclerosis of coronaries.
**Results:** A significant positive association was existed among PLR and Gensini score in the study participants (p <0.001). Regarding the diagnostic ability of PLR to forecast severe CAD, PLR is a significant predictor of severe CAD (AUC: 0.772, p <0.001). At a cut off value of >147, it has a sensitivity of 72%, specificity of 78%, PPV of 62% and NPV of 84%.
**Conclusions:** Strong positive correlation between PLR and Gensini scores was observed suggesting that PLR could be a valuable predictor for severity of coronary atherosclerosis at cutoff value of >147 with 72% sensitivity and 78% specificity that is crucial for practitioners to identify those who are most likely to develop severe CAD and who may require a more aggressive treatment strategy and intensive clinical follow-up.

**Keywords:** coronary artery disease, platelet to lymphocyte ratio, Gensini score

## Introduction
In spite advancements in the diagnosis of ischemic heart disease and its treatment, this could not change the worldwide incidence of ischemic heart disease as the most prevalent cause of mortality [1].
The procedure of Coronary atherosclerosis depends mainly on several types of factors. New markers of inflammation have been discovered that are useful as measures of the level of severity of atherosclerosis in the coronary arteries. [2].
Platelets are considered a basic supply for the mediators of inflammation [3]. Activation of platelet was demonstrated to start atherosclerosis and actually has fundamental part into its advancement [4].
Ongoing inflammatory processes cause increased synthesis of platelets via megakaryocytic series development, which leads to in relative thrombocytosis. Elevated number of platelets in the peripheral blood is usually associated with high possibility of undesired cardiac and vascular outcomes [5].
Studying lymphocytes and their impact in regulating the immunologic reaction at nearly all pathophysiologic phases of atherosclerotic process raises the level of significance of applying them as indicators of atherosclerosis [6].
The relationship between lymphocytopenia and major undesired cardiac and vascular outcomes appeared in several reviews [7, 8].

Due to a higher level of cell death, lymphocyte count greatly reduces with prolonged inflammation. Compared to other immune responses, lymphocytes are more efficient. Meanwhile, a harmful inflammatory response is produced by neutrophils. Lymphcytopenia was learned to be incredibly in a population-based study among people with determined stable coronary artery disease was associated with lower survival. (CAD). Additionally, they propose that lymphocytopenia is a potential risk factor and a free crucial prognostic marker in stable CAD [6].

But it has been determined that the number of lymphocytes is a significant early indicator of physiological "stress" and an inflammatory response to it. [6]. Previous studies showed the causal link between a number of hematologic factors, mainly the neutrophil/lymphocyte ratio, which is also and CAD. The neutrophil/lymphocyte ratio has been shown to be both prognostic and predictive in several types of cardiac and vascular disorders. [9].

This research aims to determine the significance of platelet to lymphocyte ratio (PLR) as a marker of atherosclerosis in the coronary arteries severity, to determine the relationship between PLR and CAD atherosclerosis severity, and to determine the PLR cut-off value that indicates severe CAD.

## Patients and Methods

180 patients arranged for selective diagnostic coronary angiography tests at Tanta University Hospitals' Department of Cardiology from May 2021 to May 2022 were the subject of this case control research.

All participants gave their informed permission after the research received approval from Tanta University's Faculty of Medicine's ethics committee.

Patients with significant valvular or rheumatic heart disease, haematological disease, tumours, chronic liver or renal disease, systemic inflammatory disease, acute infection, autoimmune disorders, decompensated heart failure, cardiac shock, or patients taking treatment with steroids for any reason were excluded from the study.

The participants were split into three equal groups regarding Gensini score and coronary angiography. Group 1: 60 patients with normal angiography of coronary and zero Gensini score. Group 2: 60 mild atherosclerotic patients CAD and Gensini scores of <25 points. Group 3: 60 patients with severe atherosclerotic CAD and Gensini scores of ≥ 25 to detect the value of PLR as a sign of atherosclerosis of the coronary arteries severity.

All patients were applied to these procedures: History taking, general, & local examinations, laboratory investigation: serum urea and creatinine, total and differential leukocyte numbers, blood glucose fasting level, lipid profile cardiac enzymes include serum troponin and CK-MB and complete blood count, and complete blood count including: The NLR and the LMR were assessed as the neutrophil/lymphocyte ratio and as lymphocytes count to monocytes count ratio, respectively and parameters of platelete (Count, PDW, MPV) had been identified utilizing an automated cell counter machine (Abbott Cell-Dyn 4.000, Abbott Park, IL, USA) [10], resting 12 leads ECG [11, 12], angiographic assessment and Gensini score.

All participants in the study performed diagnostic angiography. The femoral artery was punctured by the method of Seldinger and retrograde coronary angiography was done to all patients. All of the angiograms of the coronary will carefully be estimated by two experienced interventionists. The specific location of each coronary angiography and the rate of stenosis in the arteries for all lesions of the coronary artery were determined. The severity of CAD was determined by the Gensini grading method. The level and sites of coronary artery stenosis are classified and evaluated via this technique **[13]**.

The level of atherosclerosis was assessed by GS **[14]** Regarding a total score of 0 to 32, eight coronary segments' severest stenosis was scored from 1 to 4 (1%-49% luminal diameter decrease: 1 point; 50%-74% stenosis, 2 points; 75%-99% stenosis, 3 points; and 100% occlusion, 4 points). The degree of severity of coronary atherosclerosis is determined by this value. without calcification and after dissection, a filling flaw encircled by a contrast medium was described as a coronary thrombosis. Any anterograde opacification was considered to be a part of total occlusion. Any coronary calcified lesion that may be seen by angiogram is considered to have coronary calcification. To evaluate the severity of CAD, the Gensini assessment method was used.

According to the degree of narrowing in the lumen and the regional significance of each coronary stenosis, a grade of severity was given to each patient's GS depending on the coronary arteriogram. Smaller lumen diameter and eccentric plaques and concentric lesions' roentgenographic morphology were assessed (reductions of 25%, 50%, 75%, 90%, and 99% were assigned GS of 1, 2, 4, 8, 16, and 32, correspondingly).

## Statistical Analysis

The SPSS version 25 (IBM Inc., Chicago, IL, USA) was used for the statistical evaluation. In order to determine whether parametric or nonparametric statistical testing should be used the distribution of quantitative parameters was analysed utilizing the histogram and shapiro-wilks normality test

Age and other parametric variables, for example, were reported as a mean as well as a standard deviation (SD) and comparing between the 3 groups utilizing the F test, with the post hoc (Tukey) test used to evaluate each pair of groups separately. The paired T test was utilized to analyse comparisons between two variables within the same group.

The Kruskal-Wallis test was used to analyse non-parametric variables (such as the VAS), which were then further analysed using the Mann-Whitney (U) test to compare each pair of groups. Wilcoxon test was used for comparing two variables within the same group. Categorial variables, such as sex, were analysed statistically using the Chi-square test and represented as percentage and frequency. It was utilised to identify relationships between the two quantitative variables in a group using the linear correlation coefficient (r). Analysis of the Receiver Operating Characteristic curve (ROC-curve) A curve that extends from the lower left corner to the upper left corner & then to the upper right corner is regarded as the ideal test. The total diagnostic performance of each test was evaluated using ROC curve analysis. The total test performance is assessed using the under the curve area (AUC), with an under the curve area of around 100% being the best test performance and one of >50% representing satisfactory performance. Statistical significance was identified as a two-tailed P value ≤ 0.05.

## Results

Regarding the baseline features of the studied groups, a substantial variation was existed among their age (p <0.001). Age of the groups II and III was substantially higher than age of group I (p =0.006 and <0.001 respectively) no substantial variation was existed among the

age of the II and III groups. But no substantial variation in gender was existed among the studied groups. no substantial variation was existed in the indicators for risk of CAD (family history of CAD, Diabetes mellitus, and smoking) between the studied groups**.** Hypertension was substantially variance among the 3 groups. Participants with hypertension were substantially greater in group III than group I, but no substantial variation was existed among the I and II group and between II and III groups. no substantial variation in heart rate (HR) was existed among the studied groups**.** A substantial variation in systolic blood pressure (SBP) and diastolic blood pressure (DBP) was existed among the studied groups (p =0.002, 0.006 respectively)**.** SBP and DBP were substantially greater in group III than I and II groups, but no substantial variation was existed among group I and group II. (Table 1).

**Table 1:** Baseline characteristics, risk factors of CAD and clinical data of the studied groups

| | | Group I (n =60) | Group II (n =60) | Group III (n =60) | p value |
|---|---|---|---|---|---|
| Age (years) | Mean ± SD | 53.68 ± 11.89 | 60.22 ± 9.63 | 62.33 ± 12.56 | <0.001* P1:0.006* P2:<0.001* P3: 0.569 |
| Gender | Male | 33 (55%) | 39 (65%) | 42 (70%) | 0.223 |
| | Female | 27 (45%) | 21 (35%) | 18 (30%) | |
| DM | Present | 18 (30%) | 21 (35%) | 29 (48%) | 0.101 |
| | Not present | 42 (70%) | 39 (65%) | 31 (52%) | |
| HTN | Present | 21 (35%) | 22 (37%) | 33 (55%) | 0.048* P1: 1.000 P2: 0.043* P3: 0.066 |
| | Not present | 39 (65%) | 38 (63%) | 27 (45%) | |
| Family history of CAD | Present | 6 (10%) | 15 (25%) | 11 (18%) | 0.098 |
| | Not present | 54 (90%) | 45 (75%) | 49 (82%) | |
| Smoking | Smoker | 15 (25%) | 26 (43%) | 26 (43%) | 0.056 |
| | Non-smoker | 45 (75%) | 34 (57%) | 34 (57%) | |
| HR (bpm) | Mean ± SD | 66.71 ± 7.09 | 70.12 ± 9.55 | 67.58 ± 12.63 | 0.161 |
| SBP (mmHg) | Mean ± SD | 130.9 ± 20.04 | 134.6 ± 22.89 | 145.43 ± 24.73 | 0.002* P1: 0.649 P2: 0.002* P3: 0.026* |
| DBP (mmHg) | Mean ± SD | 78.15 ± 7.18 | 77.48 ± 13.14 | 83.08 ± 10.07 | 0.006* P1: 0.935 P2: 0.027* P3: 0.010* |

CAD: Coronary artery disease, DM: Diabetes mellites, MI: Myocardial infarction, HTN: Hypertension, HR: Heart rate, DBP: Diastolic blood pressure, SBP: Systolic blood pressure *Statistically significant as p value ≤ 0.05, P1: Significance between group I and group II, P2: Significance between group I and group III, P3: Significance between group II and group III.

Regarding biochemical data of the study participants, C-reactive protein was substantially different between the studied groups ($p$<0.001). C-reactive protein was substantially greater in both II and III group than group I (p=0.048, <0.001 correspondingly) but no substantial variation was existed among both groups II and III. RBG was substantially varied among the studied groups ($p$<0.001). RBG was substantially higher in both groups II and III than group I ($p$<0.001). But substantial variation in RBG was existed among both groups II and III no substantial variation in serum creatinine was existed among the studied groups no substantial variation in total cholesterol, LDL, and triglycerides was existed among the studied groups.

HDL was substantially varied among the studied groups ($p$<0.001). it was substantially decreased in group III than both groups I and II ($p$<0.001 and =0.008 correspondingly) but no substantial variation was existed among both groups II and I. Regarding hematological data of the study participants, no substantial variation in Hb, MPV, and lymphocytes was existed amog the studied groups. WBCs were substantially varied among the studied group (p <0.001) it was substantially elevated in group III than group I and group II ($p$<0.001) but no substantial variation was existed among both groups I and II. Platelets were substantially varied among the studied group ($p$<0.001) it was substantially greater in both II and III groups than group I (p=0.003 and <0.001 correspondingly) and was substantially greater in group III than group II. Lymphocytes were substantially varied among the studied group ($p$<0.003) it was substantially decreased in group III than group I and group II (p=0.003 and 0.033 correspondingly) but no substantial variation was existed among both groups I and II. PLR was substantially varied among the studied group ($p$<0.001). It was substantially greater in group III than group I and group II ($p$<0.001) and was substantially higher in group II than group I ($p$<0.001). (Table 2)

**Table 2:** Biochemical data, lipid profile and hematological investigations of the studied groups

| | | Group I (n =60) | Group II (n =60) | Group III (n =60) | p value |
|---|---|---|---|---|---|
| C-reactive protein (mg/L) | Median (IQR) | 4 (3 - 9) | 7.5 (4 - 12.25) | 10 (2.75 - 18.25) | <0.001* P1:0.048* P2:<0.001* P3:0.187 |
| RBG (mg/dL) | Mean ± SD | 143.68±47.53 | 187.07±57.99 | 205.6±56.21 | <0.001* P1:<0.001* P2:<0.001* P3:0.494 |
| Serum creatinine (mg/dL) | Mean ± SD | 0.82±0.11 | 0.84±0.09 | 0.82±0.16 | 0.347 |
| Total cholesterol (mg/dL) | Mean ± SD | 201.4±36.67 | 204.85±37.52 | 192.97±45.4 | 0.25 |
| LDL (mg/dL) | Mean ± SD | 107.97±27.78 | 112.7±27.14 | 118.72±35.97 | 0.158 |
| HDL (mg/dL) | Mean ± SD | 48.6±12.06 | 45.4±11.44 | 39.38±8.92 | <0.001* P1:0.244 P2:<0.001* P3:0.008* |
| Triglycerides (mg/dL) | Mean ± SD | 138.12±46.84 | 140.22±33.85 | 138.1±39.3 | 0.947 |
| Hb (g/dL) | Mean ± SD | 13.3±1.52 | 13.8±1.34 | 13.7±1.45 | 0.135 |
| WBCs $\times 10^3$ (cells/µL) | Mean ± SD | 7.03±2.42 | 7.33±2.63 | 9.57±3.45 | <0.001* P1:0.835 P2:<0.001* P3: <0.001* |
| Platelets $\times 10^3$ (cells/µL) | Mean ± SD | 226.28±43.82 | 250.7±41.36 | 269.72±35.29 | <0.001* P1:0.003* P2:<0.001* P3:0.028 |
| MPV (femtolitre) | Mean ± SD | 8.58±1.29 | 8.75±1.4 | 8.48±1.42 | 0.562 |
| Lymphocytes$\times 10^3$ (cells/µL) | Mean ± SD | 2.09±0.52 | 1.99±0.88 | 1.69±0.55 | 0.003* P1:0.691 P2:0.003* P3:0.033* |
| PLR | Mean ± SD | 112.18±24.99 | 141.4±43.96 | 172.85±46.25 | <0.001* P1:<0.001* P2:<0.001* P3:<0.001* |

RBG: Random blood glucose, HDL: High density lipoprotein, LDL: Low density lipoprotein, Hb: Hemoglobin, MPV: Mean platelet volume, WBCs: White blood cells, PLR: Platelet lymphocyte ration. *Statistically significant as p value ≤ 0.05, P1: Significance between group I and group II, P2: Significance between group I and group III, P3: Significance between group II and group III.

There was a substantial positive association across PLR and Gensini score among the study participants (p <0.001). (Table 3)

**Table 3:** Correlation between PLR and Gensini score in the study participants

| | Gensini score (n =180) | |
|---|---|---|
| | r | p value |
| PLR | 0.412 | <0.001* |

PLR: Platelet lymphocyte ratio. *Statistically significant as p value ≤ 0.05

Regarding PLR's capacity to diagnose and forecast severe CAD, PLR is a significant predictor of severe CAD (AUC: 0.772, p <0.001). At a cut off value of >147, it has a sensitivity of 72%, specificity of 78%, PPV of 62% and NPV of 84%. (Figure 1)

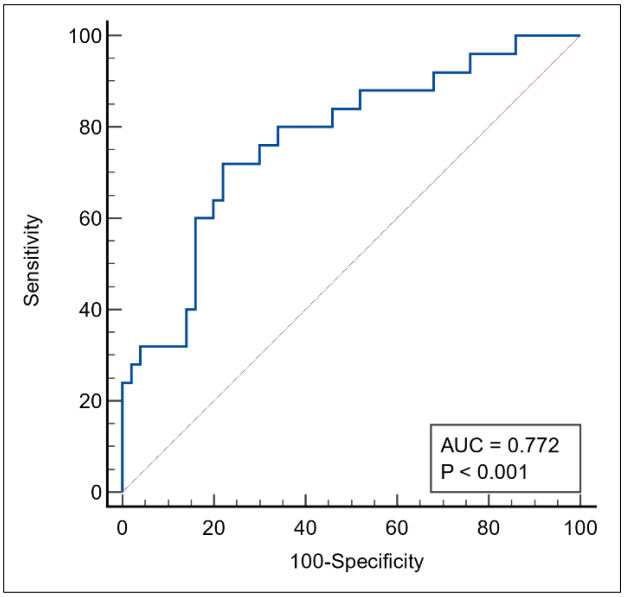


**Fig 1:** Diagnostic accuracy of PLR to predict severe CAD in the study participants

## Discussion

In individuals suffering from acute coronary syndrome, a greater PLR value became a substantial independent indicator of long-term survival, and in individuals following primary PCI, it became an independent indicator of no-reflow generation [15] The Gensini score was developed in these situations to reveal the degree and severity of coronary atherosclerosis [16].

Clinical results from the present trial indicated that no discernible change was existed in HR. The groups under study differed significantly in terms of SBP and DBP (p = 0.002 and 0.006, correspondingly). Group III had considerably greater SBP and DBP than groups I and II, but no discernible difference was existed among groups I and II.

These results are in line with that of Sari *et al*., 2015 [17], who found no statistically noteworthy variance among the control and atherosclerosis CAD groups in terms of HR (P=0.193). Contrary to our findings, the DBP was greater in the normal group contrasted to the CAD group (p = 0.005) and the SBP was not statistically different among the control and atherosclerotic CAD groups (P=0.051).

C-reactive protein was substantially varied among the studied groups in the current investigation when it came to the biochemical information of the study subjects (p <0.001). In comparison to group I, group II and group III had substantially greater levels of C-reactive protein (p = 0.048 and <0.001, correspondingly). However, no noticeable distinction was existed among groups II and III. Similar to these findings, Akboga *et al*. (2016) [18] observed that the level of CRP was substantially greater in the groups with mild to severe CAD in contrast to the control group (P> 0.01). Atherosclerosis progresses together with CRP, an accurate indicator of chronic systemic inflammation, according to Ray *et al*.'s 2010 [19] research. Additionally, CRP was shown by Danesh *et al*. in 2004 [20] to be a moderate indicator of CAD.

RBG was also substantially different amongst the groups under investigation ($p$<0.001). Contrasted to group I, RBG was considerably greater in groups II and III (p <0.001). However, no discernible variation in RBG was existed among groups II and III. Similar findings were reached by Yüksel *et al*. in 2015 [21], who found that patients with mild to severe atherosclerosis had glucose levels that were noticeably greater than that in the control group.

According to the present research, no substantial variance was existed in the tested groups' total cholesterol, LDL, or triglycerides. However, the current research found that HDL was substantially varied across the groups it was investigated in (p <0.001). no substantial variance was existed among group II and group I, however it was substantially less in group III than in groups I and II (p <0.001 and =0.008, correspondingly).

Similarly to these results, Akboga *et al*., 2016 [18] discovered that no substantial disparity was existed in total cholesterol and triglyceride levels among control subjects and moderate and severe individuals with CAD. In addition, the group with severe CAD had considerably lower HDL levels than the control group ($P$< .05. Additionally, Yüksel *et al*., 2015 [21] findings were consistent with this research; the severe atherosclerosis group had levels of HDL that were substantially smaller than controls (p < 0.001), but the mild and severe atherosclerosis groups had identical levels (p=0.137).

Lymphocytes in the present research substantially differed from the examined group (p 0.003). Compared to groups I and II, it was substantially smaller in group III (p = 0.003 and 0.033, correspondingly), while no discernible variation was existed among groups I and II. Between the study group, there were noticeably varied WBCs ($p$<0.001). Compared to groups I and II, it was considerably greater in group III (p 0.001), while no discernible variation was existed among groups I and II. According to Akboga *et al*.'s 2016 [18] research, which is consistent with these findings, the control group's lymphocyte count was considerably greater than that of the moderate and severe CAD groups (P>0.01). The results of this research are consistent with those of Yüksel *et al*. (2015) [21], who found that WBC was equivalent in the mild atherosclerosis and control groups (p=0.779) but substantially greater in the severe atherosclerosis group ($p$<0.001). Additionally, all three groups' lymphocyte counts were similar (p=0.337). In addition, Horne *et al*. 2005 [22] provided data showing that individuals with CAD who had low lymphocyte counts had a considerably higher risk of adverse outcomes.

Low lymphocyte counts were found to be strongly linked with worse outcomes for individuals with NSTEMI in Azab *et al*.'s 2012 [23] research. Additionally, they showed that a poorer prognosis was significantly correlated with both a lower lymphocyte count and a larger platelet count.

Additionally, the current research found that the analysed group's platelets were considerably different from one another ($p$<0.001). When contrasted to group I, it was considerably greater in groups II and III (p = 0.003 and <0.001, correspondingly) and greater in group III than group II.

According to these findings, Akboga *et al*. (2016) [18] found that PLT in severe CAD patients was substantially higher than in control and mild CAD patients (P > 0.01).

The current findings are consistent with those of Yüksel *et al*. in 2015 [21], who showed that although platelet counts in the last 2 groups were comparable (p=0.671), the severe atherosclerosis group had substantially greater platelet counts than the mild atherosclerosis group and controls.

Additionally, the present results demonstrated that PLR varied considerably across the groups that were being tested

(p <0.001). It was substantially greater in group III contrasted to groups I and II ($p$< 0.001) and in group II compared to group I (p <0.001).

Interestingly, Akboga *et al*., 2016 [18] also found data that were comparable, namely that PLR in severe CAD was considerably higher than in control and mild CAD groups (P > 0.01), contrasted to those groups.

Similar findings were found by Yüksel *et al*. in 2015 [21], who found that PLR was considerably greater in the group with severe atherosclerosis compared to the groups with mild atherosclerosis and controls ($p$<0.001). In addition, PLR was considerably greater in individuals with CAD in comparison to controls ($p$< 0.001).

Additionally, Yayla *et al*. (2015) [24] stated that chronic inflammation may result in a rise in PLR, and that elevated PLR may be an indicator of ongoing low-grade inflammation. Low-grade inflammation has been linked to an increased risk of a variety of conditions, that include infarct-related artery patency, slow coronary flow, and negative CV events.

Additionally, the current research examined the relationships between these indicators and found that the PLR and Gensini score in the study group had a strong positive relationship ($p$<0.001). In terms of the positive association among PLR and Gensini score as an indicator of the extent and depth of coronary atherosclerosis, the present study's results were confirmatory of Akboga *et al*., 2016 [18] ($p$<0.001). Furthermore, among individuals who had stable CAD, Yükselm, 2015 [25] revealed an independent relationship among preprocedural PLR and the extent of coronary atherosclerosis; PLR values of individuals with CAD strongly linked with their Gensini scores (r=0.268, $p$<0.001).

These results are comparable to those of Sari *et al*., 2015 [17] who revealed a substantial positive connection among PLR values and Gensini scores in individuals with CAD.

PLR is an accurate indicator of severe CAD in the current investigation, which examines the diagnostic capacity of PLR for predicting CAD (AUC: 0.772, p <0.001). It has a specificity of 78%, sensitivity of 72%, PPV of 62%, and NPV of 84% at a cutoff value of >147.

These findings are comparable to those of Akboga *et al*., 2016 [18] who found that PLR is a reliable indicator of the existence of severe coronary atherosclerosis. At a cutoff value of >109.5, PLR had a specificity of 58%, a sensitivity of 70%, a positive predictive value of 52.2%, and a negative predictive value of 74.7%.

The PLR at a cut-off level of 111 indicated severe atherosclerosis with a specificity of 59%, and a sensitivity of 61% according to Yükselm, 2015 [25]

Kurtul *et al*., 2014 [26].also included 1,016 individuals in their research who had urgent CA upon diagnosis to examine the efficacy of PLR in predicting the complexity and severity of coronary atherosclerosis among individuals with ACS. The PLR cut-off value was 116 with 71% sensitivity and 66% specificity, according to ROC curve analysis.

The research has certain drawbacks, such as the single-centric design and the limited sample size. Only coronary angiography, which only shows the coronary artery lumen and fails to offer detailed information on the load of coronary plaque, was used to assess the coronary atherosclerosis. Other well-known inflammatory indicators including interleukin-6 and tumour necrosis factor-a were absent from the research. The relationship among PLR and CAD severity requires more prospective research with bigger sample sizes. To give more precise data on the degree of coronary atherosclerosis, multi-centric prospective studies using intravascular ultrasonography and/or coronary CT are required. PLR may serve as a useful indicator of the extent of coronary atherosclerosis.

## Conclusions

In CAD individuals with atherosclerosis, platelets count and PLR were substantially greater in CAD individuals with Gensini scores of ≥ 25 contrasted to the control group. Moreover, strong positive association among PLR and Gensini scores was observed suggesting that PLR could be a valuable predictor for severity of coronary atherosclerosis at cutoff value of >147 with 72% sensitivity and 78% specificity that is important for practitioners in determining persons who may require more aggressive therapy strategy and tighter clinical follow-up because they are at high risk for developing advanced CAD.

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**Conflict of Interest:** Nil

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