Correlation between Complete Blood Count Parameters and Severity of Coronary Atherosclerosis in Patients with Acute Coronary Syndrome Detected by Coronary Angiography

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Abstract

Background: Acute coronary syndromes (ACS), including STEMI, NSTEMI, and unstable angina, involve reduced blood supply to the heart. Inflammatory markers like leukocytosis and CRP contribute to ACS and atherosclerosis. Complete cell count (CBC) parameters predict long-term outcomes, and CT coronary angiography is useful for low-risk chest pain patients.

Objectives: To examine the correlation between CBC parameters and coronary artery disease severity in ACS patients using coronary angiography.

Patients and methods: This cross-sectional study at Qena University Hospitals included 100 ACS patients with atherosclerosis aged ≥18. Exclusion criteria included chronic coronary syndrome and hematological or immunological disorders. CBC, lipid profile, HbA1c, and renal function tests were collected. Coronary angiography assessed severity using the Gensini score, classifying patients into mild (1-29) or severe (≥30) atherosclerosis.

Results: The mean age 47.3 ± 7.29 years. 71% were males. Mean BMI was 29.38 ± 5.02 Kg/m^2. 29% had diabetes, 25% had hypertension, and 29% were smokers. The mean Gensini score was 61.72 ± 43.28; 34% had mild and 66% had severe atherosclerosis. Severe cases had higher BMI, hypertension, RDW, WBC count, platelet indices, NLR, cholesterol, triglycerides, LDL, FBG, HbA1c, urea, and creatinine ($P < 0.0001$ for all). The Gensini score correlated with BMI, RDW, cholesterol, triglycerides, FBG, HbA1c, and MPV ($P < 0.0001$ for all).

Conclusion: Elevated RDW, WBC count, NLR, MPV, PDW, lipid levels, poor glycemic control, and renal impairment markers were linked to severe atherosclerosis in ACS patients.

Keywords: Complete blood count; Coronary atherosclerosis; Acute coronary syndrome; Coronary angiography.

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Introduction

ACS involves a sudden reduction in blood supply to the heart and encompasses STEMI, NSTEMI, and unstable angina. The most common symptom of ACS is chest discomfort at rest, although many patients present with nonspecific symptoms such as dyspnea **(Zeitz et al., 2022; Khurshid et al., 2023).**

Inflammatory markers, including leukocytosis and elevated C-reactive protein (CRP), play a critical role in the pathophysiology of ACS and atherosclerosis by contributing to endothelial dysfunction and other pathological mechanisms. Complete blood count (CBC) parameters have demonstrated their utility in predicting long-term outcomes in ACS patients. Additionally, computed tomography (CT) coronary angiography has proven effective in expediting care for low-risk patients presenting with chest pain. For patients with suspected ACS who are at low or intermediate risk, current guidelines recommend further testing and observation to ensure accurate diagnosis and appropriate management **(Bhatt et al., 2022).**

This study aims to examine the correlation between CBC parameters and the severity of coronary artery disease in ACS patients using coronary angiography.

Patients and methods

This cross-sectional study was conducted in the Internal Medicine Department at Qena University Hospitals for over eight months. The study included 100 patients with atherosclerosis who met the inclusion criteria: individuals aged ≥18 years with acute coronary syndrome undergoing coronary angiography. Patients with chronic coronary syndrome, hematological or immunological disorders, infections, respiratory disorders, liver or renal disease, or malignancy were excluded.

Sample size calculation: the sample size was calculated using a standard formula, considering 5% type 1 error ($P < 0.05$) with a Z-value of 1.96. The sample size was calculated using the following formula:

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

Where:

- $Z_{\alpha/2}$ = critical value for the significance level α (e.g., 1.96 for 5% significance)
- Z_{β} = critical value for statistical power $1-\beta$ (e.g., 0.84 for 80% power)
- C = standardized effect size = 0.34 **(Uysal et al., 2016).**

The calculated sample size using epi-info was 68 and was increased to 100 to avoid the risk of drop out during follow-up.

Patients were selected from the coronary care unit (CCU) and outpatient cardiac clinic at Qena University Hospitals, ensuring adherence to the inclusion and exclusion criteria.

Laboratory Investigations

Serum Preparation

After an overnight fast, venous blood samples (5 mL) were collected using EDTA and basic tubes. To get serum, blood in standard tubes was centrifuged at 3000×g for 10 minutes after being allowed to coagulate for 30 minutes at 37°C.

Investigations

- **Fasting blood sugar (FBG)** was measured using Accu-chek active glucometer (Roche Diagnostic Corporation, Switzerland).
- **Complete Blood Count (CBC):** Parameters such as RBC count, hemoglobin, RDW, WBC count, neutrophil-to-lymphocyte ratio (NLR), PLT and platelet to lymphocyte ratio (PLR) were analyzed. Platelet indices, including MPV, PCT, and PDW, were assessed using an automated cell counter (Cell-Dyne Ruby, Abbott Diagnostics).
- **Lipid Profile:** Total cholesterol, High density lipoprotein (HDL), and triglyceride (TG) levels were measured using a Cobas Integra-400 plus analyzer (Roche

Diagnostics corporation, Switzerland), while low density lipoprotein (LDL) was calculated using Friedewald's formula.

- **Glycosylated Hemoglobin (HbA1c):** Measured using HPLC with a Variant II Turbo system (Bio-Rad Laboratories corporation, USA).
- **Renal Function Tests:**
- **Serum Creatinine** was Measured via Jaffe's reaction using Roche Cobas with Creatinine Jaffe (CREJ2) (Roche Diagnostics corporation, Switzerland).
- **Blood Urea Nitrogen (BUN)** was also measured suing Roche Cobas for Urea (Roche Diagnostics corporation, Switzerland).

Coronary Angiography

Procedure

We conducted a coronary angiography using Innova IGS 520 (GE, USA) through the right femoral approach using a 6F catheter. Imaging was conducted in different views, analyzing vessels with diameters ≥1.5 mm, even those remote from full occlusions.

Coronary Artery Segmentation

Coronary arteries were segmented per American Heart Association guidelines **(Leipsic et al., 2014):**

- **Right Coronary Artery (RCA):** Proximal, middle, and distal parts.
- **Posterior Descending Artery:** Independent segment due to variable origin.
- **Left Circumflex Artery (LCX):** Proximal and distal parts.
- **Left Anterior Descending Artery (LAD):** Proximal, middle, and distal parts.
- **Other Branches:** Diagonal (DIA), obtuse marginal (OM), and posterolateral (PLA) branches were considered independent segments.

***Severity Assessment Using Gensini Score (GS)* (Kashani et al., 2016):**

The severity of coronary artery disease was assessed using the Gensini score (GS), which quantified the degree of luminal stenosis. Stenosis was graded as follows: 1%-25% narrowing received 1 point, 26%-50% received 2 points, 51%-75% received 4 points, 76%-90% received 8 points, 91%-99% received 16 points, and complete stenosis received 32 points. These scores were further adjusted using vessel-specific coefficients to account for the importance of the affected vessel. The left main coronary artery was assigned a coefficient of 5, the LAD artery and the proximal LCX were assigned a coefficient of 2.5, and the proximal RCA was assigned a coefficient of 1.

Ethical approval code

- All patients provided informed consent before participation. A comprehensive history was taken, followed by a general physical examination.
- **Ethical approval code: SVU-MED-MED018-1-23-8-701.**

The total Gensini score for each patient was done by summing the stenosis scores multiplied by their respective coefficients. Based on their total scores, patients fell into one of two categories: those with scores between 1 and 29 were categorized as having mild atherosclerosis, while those with scores of 30 or higher were categorized as having severe atherosclerosis.

Statistical analysis

SPSS version 20 was used for statistical analysis. Quantitative data were shown as mean ± standard deviation (SD), whereas qualitative data were expressed as numbers and percentages. Shapiro-Wilk Test was used to assess data normality. The Mann-Whitney test was used to compare two independent groups. The chi-square test was used to compare categorical variables. The relationships between the variables were examined using Pearson correlation. Statistical significance was defined as a p-value of less than 0.05.

Results

The study 100 cases had a mean age of 47.3 ± 7.29 years, with 71% males. Demographic data was demonstrated in **Table 1.**

BMI was significantly higher, and hypertension was more prevalent in severe atherosclerosis group. The rest of the demographic data showed non-significant differences. **Table 2.**

RDW, WBCs, NLR, MPV, PDW, cholesterol levels, triglycerides, LDL, FBG, HbA1c, serum creatinine and urea were significantly higher in the severe atherosclerosis group. While Plataeltsa md PLR were significantly lower in the atherosclerosis group. **Table 3.**

Table 1. Demographic Data among all included subjects in our study

	Value (N = 100)
Demographic Data	
Age (mean ± SD)	47.3 ± 7.29
Sex N (%)	
• Male	71 (71%)
• Female	29 (29%)
BMI (mean ± SD)	29.38 ± 5.02
Diabetes Mellitus	29 (29%)
Duration of diabetes (months) (mean ± SD)	49.08 ± 16.94
Hypertension	25 (25%)
Smoking N (%)	29 (29%)
Gensini score (mean ± SD)	61.72 ± 43.28
• Mild atherosclerosis N (%)	34 (34%)
• Severe atherosclerosis N (%)	66 (66%)

BMI: Body Mass Index.

Table 2. Comparison between cases with mild and severe atherosclerosis regarding Demographic Data

Variables	**Mild atherosclerosis (N = 34)**	**Severe atherosclerosis (N = 66)**	**P-Value**
Demographic Data			
Age	46.91 ± 6.31	47.5 ± 7.74	0.7057 [MWU]
Sex			
Male	23 (67.65%)	48 (72.73%)	0.6002 [X]
Female	11 (32.35%)	18 (27.27%)	
BMI	26.13 ± 2.72	31.05 ± 5.11	<0.0001* [MWU]
Diabetes Mellitus	0 (0%)	29 (43.94%)	
Duration of diabetes (months)		49.08 ± 16.94	
Hypertension	3 (8.82%)	22 (33.33%)	0.007* [X]
Smoking	9 (26.47%)	20 (30.3%)	0.6927 [X]

BMI: Body Mass Index. MWU: Mann Whitney U-test, X: chi square test.

Table 3. Lab data comparison between cases with mild and severe atherosclerosis.

Variables	**Mild atherosclerosis (N = 34)**	**Severe atherosclerosis (N = 66)**	**P-Value**
CBC			

Hb	18 ± 21.15	16.08 ± 15.33	0.6081 [MWU]
RDW	13.74 ± 0.46	14.46 ± 0.31	<0.0001* [MWU]
Platelets	227.79 ± 46.08	183.94 ± 38.18	<0.0001* [MWU]
WBCs	6.51 ± 0.35	7.05 ± 0.31	<0.0001* [MWU]
NLR	2.51 ± 0.14	2.75 ± 0.18	<0.0001* [MWU]
PLR	107.58 ± 23.15	87.8 ± 18.21	<0.0001* [MWU]
Platelets Indices			
MPV	8 ± 0.31	10.56 ± 1.7	<0.0001* [MWU]
PCT	0.22 ± 0.01	0.22 ± 0.01	0.9107 [MWU]
PDW	13.88 ± 2.01	18 ± 3.42	<0.0001* [MWU]
Lipid Profile			
Cholesterol	134.88 ± 34.31	199.15 ± 64.68	<0.0001* [MWU]
Triglycerides	128 ± 63.9	204.83 ± 100.65	0.0001* [MWU]
HDL	41.18 ± 7.76	40.03 ± 7.15	0.4673 [MWU]
LDL	48.38 ± 9.11	94.88 ± 42.18	<0.0001* [MWU]
Glycemic Control			
FBG	90.56 ± 7.7	179.44 ± 53.14	<0.0001* [MWU]
HbA1c	5.39 ± 0.33	7.57 ± 1.51	<0.0001* [MWU]
Renal Function			
Serum Urea	20.62 ± 3.95	35.06 ± 13.54	<0.0001* [MWU]
Serum Creatinine	0.88 ± 0.16	1.16 ± 0.38	0.0001* [MWU]

MWU: Mann Whitney U-test.

Hb: Hemoglobin, RDW: Red Cell Distribution Width, WBCs: White Blood Cells, NLR: Neutrophil-to-Lymphocyte ratio, CBC: Complete Blood Count. Cholesterol: Total Cholesterol, HDL: High-Density Lipoprotein, LDL: Low-Density Lipoprotein. FBG: Fasting Blood Glucose, HbA1c: Glycated Hemoglobin. MPV: Mean Platelet Volume, PCT: Plateletcrit, PDW: Platelet Distribution Width.

The Gensini score correlated positively with BMI ($r = 0.461$, $P < 0.0001$), RDW ($r = 0.489$, $P < 0.0001$), WBC ($r = 0.440$, $P < 0.0001$), NLR ($r = 0.396$, $P < 0.0001$), cholesterol ($r = 0.443$, $P < 0.0001$), triglycerides ($r = 0.415$, $P < 0.0001$), LDL ($r = 0.464$, $P < 0.0001$), FBG ($r = 0.610$, $P < 0.0001$), HbA1c ($r = 0.542$, $P < 0.0001$), serum urea ($r = 0.449$, $P < 0.0001$), MPV ($r = 0.624$, $P < 0.0001$), and PDW ($r = 0.529$, $P < 0.0001$). Negative correlations were found with platelet count ($r = -0.448$, $P < 0.0001$) and PLR ($r = -0.421$, $P < 0.0001$). **Table 4, Fig 1.**

Table 4. Correlation between Gensini score with different parameters

Variables	**Gensini score**	
	r	**P-Value**
Demographic Data		
Age	0.097	0.339
BMI	.461**	<0.0001*
Laboratory Data		
CBC		
Hb	-0.061	0.549
RDW	0.489	<0.0001*
Platelets	-.448**	<0.0001*
WBCs	0.440	<0.0001*

NLR	0.396	<0.0001*
PLR	-0.421	<0.0001*
Lipid Profile		
Cholesterol	.443**	<0.0001*
Triglycerides	.415**	<0.0001*
HDL	-0.017	0.863
LDL	.464**	<0.0001*
Glycemic Control		
FBG	.610**	<0.0001*
HbA1c	.542**	<0.0001*
Renal Function		
Serum Urea	.449**	<0.0001*
Serum Creatinine	.304**	0.002*
Platelets Indices		
MPV	.624**	<0.0001*
PCT	-0.028	0.779
PDW	.529**	<0.0001*

r: Pearson correlation.

BMI: Body Mass Index, CBC: Complete Blood Count, Hb: Hemoglobin, RDW: Red Cell Distribution Width, WBCs: White Blood Cells, NLR: Neutrophil-to-Lymphocyte ratio, PLR: Platelets-to-Lymphocyte ratio HDL: High-Density Lipoprotein, LDL: Low-Density Lipoprotein, FBG: Fasting Blood Glucose, HbA1c: Glycated Hemoglobin, MPV: Mean Platelet Volume, PCT: Plateletcrit, PDW: Platelet Distribution Width.

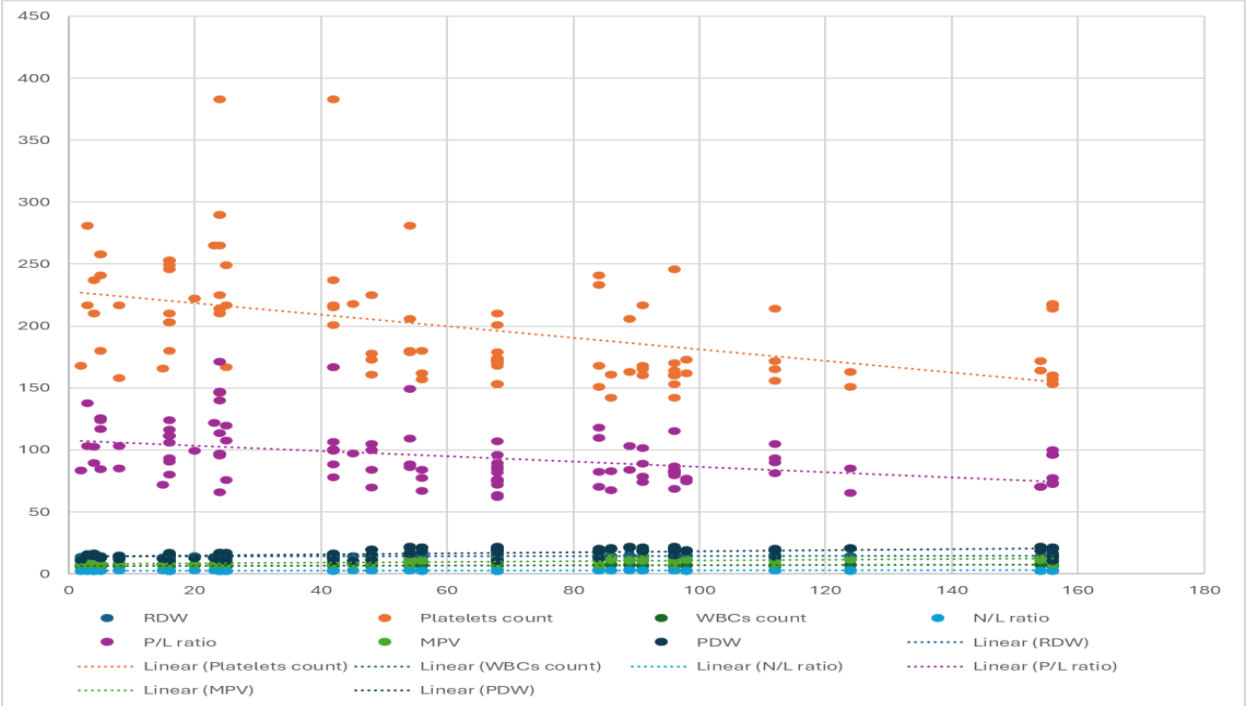

Fig 1. Correlation between Gensini score with different parameters.

Discussion

One hundred patients with ACS undergoing coronary angiography were included in this cross-sectional study, which was conducted in the internal medicine department of Qena University Hospitals. The participants had a mean age of 47.3 ± 7.29 years, with 71% being male and a mean BMI of 29.38 ± 5.02

Kg/m^2. Among them, 29% had diabetes, 25% had hypertension, and 29% were smokers. The study found no significant age or sex differences between mild (N = 34) and severe (N = 66) atherosclerosis, but 43.94% of severe cases had diabetes. Severe atherosclerosis was associated with a significantly higher BMI and higher hypertension prevalence (P = 0.007).

Our study's findings align with the understanding that obesity and hypertension are key contributors to severe atherosclerosis. Obesity triggers systemic inflammation and endothelial dysfunction, leading to plaque formation, while hypertension causes endothelial damage and vascular remodeling, worsening atherosclerosis **(Henning 2021; Totoń-Żurańska et al. 2024).** These factors likely contribute to the higher prevalence of severe atherosclerosis in our study population. In support of our study, **Hindieh et al. (2016)** included 763 patients with premature ACS, reporting a median age of 50 years, with 30.8% women. Their cohort had 56.3% hyperlipidemia, 16.4% diabetes, 47.8% hypertension, and 42.7% current smokers.

The mean Gensini score in our study was 61.72 ± 43.28, with 34% having mild atherosclerosis and 66% having severe atherosclerosis. This is consistent with **Van Hoang et al. (2021),** who found that Gensini scores in the low, intermediate, and high-risk groups of acute coronary syndrome patients were 11.8 ± 11.5, 27.4 ± 30.9, and 42.9 ± 29.7, respectively. Additionally, Turan et al. (2015) reported a Gensini score of 70.4 ± 35.9 in their study on oxidative stress markers in acute coronary syndrome patients, and **Cakar et al. (2014)** found Gensini scores of 26 ± 29, 29 ± 19, and 38 ± 23 in the categories of low, moderate, and high risk, respectively, with significant differences between the two groups (p = 0.013).

RDW, WBCs, NLR, MPV, PDW were significantly higher in the severe atherosclerosis group. While platelet count and PLR were significantly lower in the atherosclerosis group. These findings suggest that increased inflammation and vascular stress are associated with severe atherosclerosis, as elevated RDW reflects chronic inflammation, and higher WBC and NLR indicate heightened immune activity contributing to plaque instability and vascular damage **(Vayá et al. 2015; Balta et al. 2016).** Additionally, the lower platelet count in severe cases is likely due to increased platelet activation and consumption during plaque formation and thrombosis **(Koupenova et al. 2017).**

The correlation analysis revealed significant findings, with BMI positively correlating with the Gensini score (r = 0.461, P < 0.0001). RDW (r = 0.489, P < 0.0001), WBCs (r = 0.440, P < 0.0001), and NLR (r = 0.396, P < 0.0001) showed positive correlations, while platelets (r = -0.448, P < 0.0001) and PLR (r = -0.421, P < 0.0001) had negative correlations. Glycemic control (FBG: r = 0.610, P < 0.0001; HbA1c: r = 0.542, P < 0.0001) and renal markers (urea: r = 0.449, P < 0.0001; creatinine: r = 0.304, P = 0.002) were positively correlated. MPV (r = 0.624, P < 0.0001) and PDW (r = 0.529, P < 0.0001) also had positive correlations, while PCT showed no correlation (r = -0.028, P = 0.779).

In line with our study, **Bekler et al. (2015)** investigated the relationship between PDW and the severity of CAD in ACS patients, finding a significantly higher neutrophil-lymphocyte ratio (3.1 vs. 2.5, P = 0.012) and lower baseline platelet counts (220 vs. 233 103/mm3, P = 0.022) in the high PDW group. An increased PDW (>17%) was associated with more severe CAD in ACS patients. Our study also examined lipid profiles, with cholesterol averaging 177.3 ± 63.94 mg/dL, triglycerides at 178.71 ± 96.95 mg/dL, HDL at 40.42 ± 7.39 mg/dL, and LDL at 79.07 ± 41.08 mg/dL. FBG averaged 149.22 ± 60.47 mg/dL, and HbA1c was 6.83 ± 1.61%. In

severe atherosclerosis, cholesterol (199.15 ± 64.68 vs. 134.88 ± 34.31 mg/dL; P < 0.0001), triglycerides (204.83 ± 100.65 vs. 128 ± 63.9 mg/dL; P = 0.0001), and LDL (94.88 ± 42.18 vs. 48.38 ± 9.11 mg/dL; P < 0.0001) were significantly higher, while HDL levels were similar.

These lipid profile differences highlight the role of elevated LDL and triglycerides in promoting plaque formation and arterial damage, as high LDL levels contribute to cholesterol deposition in arterial walls, and elevated triglycerides exacerbate inflammation and endothelial dysfunction **(Welty, 2013; Libby, 2021).** In agreement with our study, **Amin et al. (2014)** found that a high TG/HDL-C ratio correlates with severe coronary artery disease. **Wan et al. (2015)** also found that a higher TG/HDL-C ratio is associated with all-cause mortality in ACS patients after coronary revascularization. **Greco et al. (2010)** demonstrated that patients with severe CAD had significantly higher levels of oxLDL/β2GPI and a higher rate of adverse events, further supporting the relationship between lipid profile abnormalities and CAD severity.

In severe atherosclerosis, FBG and HbA1c were significantly higher compared to mild cases. Renal function markers, including serum urea and serum creatinine P = 0.0001), were also elevated in the severe group. Chronic hyperglycemia contributes to endothelial dysfunction, inflammation, and oxidative stress, which foster plaque formation and atherosclerosis **(Yuan et al., 2019).** Similarly, elevated urea and creatinine indicate impaired renal function, which exacerbates vascular damage through hypertension and systemic inflammation **(Podkowińska and Formanowicz, 2020).**

In severe atherosclerosis, MPV was significantly higher (P < 0.0001) and PDW was significantly increased (P < 0.0001) compared to mild cases. Elevated MPV reflects increased platelet activation and the presence of larger, more reactive platelets, which enhance thrombus formation and promote plaque instability **(Korniluk et al., 2019).** Additionally, higher PDW suggests increased platelet turnover in response to inflammation, which exacerbates atherosclerosis by increasing thrombotic risk and vascular damage **(Adam et al., 2015).** These findings are consistent with those of **Gurses et al. (2014),** who reported higher MPV in patients with critical stenosis. Furthermore, **Vogiatzis et al. (2019)** discovered that there was a link between MPV and the SYNTAX score in ACS patients, supporting MPV as a potential risk stratification tool. **Murat et al. (2013)** also observed that high MPV levels were associated with more severe CAD, corroborating the importance of MPV in assessing atherosclerosis severity.

Conclusion

In conclusion, our study demonstrates a significant correlation between certain CBC parameters and the severity of CAD in patients with ACS, as determined by coronary angiography. Specifically, elevated RDW, WBC count, NLR, MPV, and PDW were associated with more severe atherosclerosis, alongside increased lipid levels, poor glycemic control, and renal impairment markers.

Limitations

The small sample size of 100 patients in this single-center study may have limited the findings' applicability to larger populations. The cross-sectional design prevents the establishment of causality between CBC parameters and coronary artery disease severity. We also did not account for potential confounding variables, such as medication use, lifestyle factors, or comorbidities beyond diabetes and hypertension, which may influence CBC parameters and atherosclerosis progression.

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