Hussein *et. al.,* Babcock Univ. Med. J.2026 9(S1):363-370
https://doi.org/10.38029/babcockuniv.med.j..v9iS1.1967
eISSN: 2756-4657

RESEARCH ARTICLE OPEN ACCESS

Association of red cell distribution width and platelet distribution width with angiographic severity of coronary artery disease in patients with stable angina: a cross-sectional study

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Submitted: 14th June 2026
Accepted: 15th October 2026
Published: 31st October 2026
ID: Orcid ID

Abstract
Objective: To assess the associations of red cell distribution width-standard deviation (RDW-SD) and platelet distribution width (PDW) with angiographic coronary artery disease (CAD) severity, using the Gensini score, in adults undergoing coronary angiography for stable angina.
Methods: This single-centre cross-sectional study recruited 65 consecutive adults who underwent coronary angiography for stable angina at Mosul Cardiac Centre, Iraq, between January and August 2025. A Gensini score ≥32 defined severe CAD. Clinical and laboratory measurements, including RDW-SD and PDW, were compared by severity. Spearman correlation, logistic regression and receiver operating characteristic (ROC) analyses were performed.
Results: Severe CAD occurred in 29/65 patients (44.6%); 36 (55.4%) had mild-to-moderate disease. RDW-SD was higher in severe CAD (45.85 ± 6.36 fL) than in mild-to-moderate disease (42.41 ± 3.25 fL; p = 0.003). RDW-SD correlated with Gensini score (rs = 0.358; p = 0.003) and remained associated after adjustment (adjusted OR, 1.320 per unit; 95% CI, 1.055–1.651; p = 0.015). RDW-SD AUC was 0.710 (95% CI, 0.582–0.837). PDW did not differ (15.60 ± 1.50 vs 15.80 ± 1.30 fL; p = 0.452) and showed poor discrimination (AUC, 0.430). Severe CAD also had higher fasting glucose and white blood cell count.
Conclusions: In this single-centre study of stable angina, RDW-SD was related to the severity of CAD as defined by Gensini but not PDW in this sample. The discrimination of RDW-SD was limited-to-moderate. The preliminary results of this study need to be confirmed in a larger population before use in the clinic.

Keywords: Coronary artery disease, Gensini score, Red cell distribution width, Stable angina, Platelet distribution width

Plain English Summary

Coronary artery disease develops when fatty deposits narrow the blood vessels supplying the heart. This study examined whether two measurements from routine blood tests, red cell distribution width (RDW-SD) and platelet distribution width (PDW), were associated with the extent of coronary artery disease in 65 patients in Iraq with stable angina. Patients with more extensive disease, as determined by coronary angiography, had higher average RDW-SD values, and RDW-SD tended to increase with the angiographic disease score. PDW did not show a clear relationship with disease severity. However, RDW-SD was only moderately effective at distinguishing patients with severe disease from those with less extensive disease. The findings suggest a possible association between RDW-SD and coronary artery disease burden, but the small, single-centre study cannot establish whether the measurement

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provides meaningful additional information beyond established clinical assessment. Larger studies are needed before any clinical application is considered.

Introduction

Coronary artery disease (CAD) remains a leading contributor to cardiovascular morbidity and mortality worldwide, with a particularly substantial impact in low- and middle-income countries where access to advanced diagnostic resources may be limited (1). In Iraq, the burden of CAD is compounded by a high prevalence of modifiable risk factors, including tobacco use, diabetes mellitus, hypertension and shifting dietary patterns (2, 3). Identifying inexpensive and readily available markers of disease burden could therefore assist clinical assessment in settings where healthcare resources are constrained. Similar challenges are encountered in other low-resource settings; in Nigeria, for example, patients undergoing coronary angiography for suspected CAD often present with advanced disease (4).

Stable angina typically reflects fixed atherosclerotic narrowing that limits coronary blood flow when myocardial oxygen demand increases. Coronary angiography remains the reference standard for establishing the distribution and severity of luminal disease. The Gensini scoring system converts the percentage of stenosis and the anatomical importance of each lesion into a composite numerical measure, allowing overall coronary involvement to be quantified (5). This approach has been widely used in cardiovascular research and provides a semi-quantitative assessment of angiographic disease burden.

Modern full blood count analysers generate indices beyond simple cell counts. Red cell distribution width-standard deviation (RDW-SD) quantifies the dispersion of erythrocyte volume and has been associated with inflammation, oxidative stress, disordered erythropoiesis and nutritional deficiencies (6, 7). These processes also participate in the pathogenesis of atherosclerosis, providing a plausible biological rationale for reported relationships between RDW and adverse cardiovascular outcomes (8, 9). However, much of the existing evidence derives from acute coronary syndrome populations or from prognostic studies, and the relevance of RDW-SD to angiographic disease extent in clinically stable patients remains uncertain.

Platelet distribution width (PDW) reflects heterogeneity in platelet volume and is sometimes interpreted as a surrogate marker of platelet activation (10, 11). Published studies have not reached consensus on whether either PDW or RDW reliably reflects angiographic CAD severity, and evidence derived specifically from stable angina populations in Iraq is limited (12, 13). The present study was therefore undertaken to assess the associations of RDW-SD and PDW with angiographic CAD severity, as measured by the Gensini score, among adults undergoing coronary angiography for stable angina at a single cardiac centre in Iraq. The primary objective was to determine whether higher values of either index accompany greater angiographic disease burden. A secondary objective was to evaluate the discriminatory performance of these indices for identifying severe CAD.

Materials and Methods

Study Design and Setting

This single-centre cross-sectional study was conducted at Mosul Cardiac Centre, Mosul, Iraq. Consecutive adults scheduled for diagnostic coronary angiography because of stable angina were recruited between January and August 2025.

Study Population and Eligibility Criteria

Participants were eligible for inclusion if stable angina was the clinical indication for coronary angiography during the study period. Stable angina was defined operationally as exertional chest discomfort relieved by rest or nitrates, with no documented episode of acute coronary syndrome within the preceding three months. Exclusion criteria comprised acute coronary syndrome, previous percutaneous coronary intervention or coronary artery bypass grafting, major valvular heart disease, haematological disorders, renal or hepatic failure, active infection, chronic inflammatory disease and malignancy. These exclusions were applied to reduce the influence of conditions known to affect haematological indices independently of coronary atherosclerosis.

Sampling and Sample Size

All eligible patients who underwent coronary angiography during the recruitment period were approached for participation. No formal a priori sample size calculation was performed; the study sample was determined by the number of eligible patients presenting during the fixed recruitment window. This limitation is acknowledged in the discussion.

Data Collection and Clinical Variables

Clinical information was obtained through structured interviews supplemented by review of the medical record. The following variables were recorded: age, sex, smoking status, hypertension, diabetes mellitus, dyslipidaemia,

family history of premature CAD, obesity and current medication use. Hypertension and diabetes mellitus were identified from established physician diagnoses or ongoing pharmacological therapy. Dyslipidaemia was defined by documented diagnosis or current lipid-lowering treatment. Obesity was defined as a body mass index of 30 kg/m² or greater. Smoking status was classified as current smoker versus non-smoker or former smoker.

Laboratory Measurements
Peripheral venous blood was drawn before angiography. The available laboratory variables comprised haemoglobin, fasting blood glucose, total cholesterol, triglycerides, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, very-low-density lipoprotein (VLDL) cholesterol, white blood cell count, platelet count, RDW-SD and PDW. All measurements were performed in the central laboratory of Mosul Cardiac Centre using the institution's standard analysers and procedures. The specific analyser model, manufacturer and quality-control procedures were not documented in the study records; this limitation is acknowledged in the discussion.

Coronary Angiography and Severity Assessment
Coronary angiograms were acquired using the Judkins approach. Two experienced cardiologists, blinded to the haematological measurements, independently reviewed each examination. Discrepancies in interpretation were resolved through joint review and consensus.
The angiographic extent of CAD was calculated using the Gensini method.[6] Stenoses of 25%, 50%, 75%, 90% and 99% were assigned 1, 2, 4, 8 and 16 points, respectively, and complete occlusion was assigned 32 points. The score for each stenosis was then weighted according to lesion location: 5 for left main disease; 2.5 for proximal left anterior descending (LAD) or proximal left circumflex (LCX) disease; 1.5 for mid-LAD disease; 1 for distal LAD, mid or distal LCX, posterior descending artery, or proximal or mid right coronary artery disease; and 0.5 for smaller branches. The sum of all weighted lesion scores constituted the patient-level Gensini score.
Patients with a Gensini score ≥ 32 were grouped into severe CAD (Group I) and patients with a Gensini score < 32 into mild-to-moderate CAD (Group II) for analysis. The threshold was chosen to be a pre-defined analytical threshold (32 is the unweighted score for complete occlusion, with location weighting to get the Gensini final score). It was used to help identify patients with significant disease burden. The threshold was determined prior to data analysis. The interobserver agreement for the Gensini scoring was not formally quantified before consensus review – this is acknowledged.

Statistical Analysis
IBM SPSS Statistics version 29.0 was used for all analyses. Distributional form was examined using the Shapiro-Wilk test. Continuous variables were summarised as mean ± standard deviation when approximately normally distributed and as median with interquartile range otherwise. Depending on distribution, groups were compared using an independent-samples t-test or the Mann-Whitney U test. Categorical variables were described as frequency and percentage and compared using Pearson's chi-square test or Fisher's exact test, as appropriate. Associations between each blood-cell index and the continuous Gensini score were estimated using Spearman correlation. Severe CAD served as the dependent outcome in a binary logistic regression model that included covariates selected on clinical grounds: hypertension, diabetes mellitus, LDL cholesterol, white blood cell count, platelet count, RDW-SD and PDW. These variables were chosen because they represent established cardiovascular risk factors or potential confounders of the relationship between haematological indices and CAD severity. Adjusted odds ratios were reported with 95% confidence intervals and p-values.
A receiver operating characteristic (ROC) curve was generated to measure the performance of RDW-SD and PDW in determining the ability to correctly categorise the patients as above or below the severe-CAD threshold. The area under the curve (AUC) with 95% confidence interval (CI) was determined. A statistical test was performed, and $p < 0.05$ indicated statistical significance. All inferential findings should be viewed as exploratory, as multiple comparisons were not performed with multiplicity adjustment.

Results
Participant Characteristics
We included 65 patients with stable angina in the analysis. Gensini scoring classified 29 patients (44.6%) as having severe CAD and 36 patients (55.4%) as having mild-to-moderate CAD.
The mean age of the severe CAD group was 60.10 ± 9.79 years, compared with 57.83 ± 10.17 years in the mild-to-moderate group; this difference was not statistically significant ($p = 0.365$). Men represented 79.3% of the severe CAD group compared with 50.0% of the mild-to-moderate group, a difference that reached statistical significance ($p = 0.015$). The two groups had comparable prevalences of hypertension, diabetes mellitus, dyslipidaemia,

obesity and family history of CAD. Current smoking was numerically more common in the severe CAD group (41.4% vs 19.4%), although this comparison did not reach the conventional threshold for statistical significance (p = 0.053). Baseline demographic and clinical characteristics are presented in Table 1.

Table 1: Demographic and clinical profile stratified by angiographic CAD severity

Characteristic	Severe CAD (n = 29)	Mild-to-moderate CAD (n = 36)	p-value
Age (years)	60.10 ± 9.79	57.83 ± 10.17	0.365[a]
Sex			0.015[b]
Male	23 (79.3%)	18 (50.0%)	
Female	6 (20.7%)	18 (50.0%)	
Hypertension	20 (69.0%)	25 (69.4%)	0.967[b]
Diabetes mellitus	18 (62.1%)	15 (41.7%)	0.102[b]
Dyslipidaemia	21 (72.4%)	23 (63.9%)	0.465[b]
Smoking	12 (41.4%)	7 (19.4%)	0.053[b]
Family history of CAD	6 (20.7%)	5 (13.9%)	0.345[c]
Obesity	11 (37.9%)	16 (44.4%)	0.596[b]

Values are presented as mean ± standard deviation or frequency (percentage). CAD, coronary artery disease; [a] Independent-samples t-test; [b] Pearson's chi-square test; [c] Fisher's exact test

Laboratory Measurements

Laboratory comparisons between the two severity groups are presented in Table 2. Severe CAD was accompanied by significantly higher fasting blood glucose (145.04 ± 50.15 vs 126.47 ± 51.40 mg/dL; p = 0.038) and white blood cell count (8.71 ± 2.66 vs 7.15 ± 2.11 × 10^9/L; p = 0.005). RDW-SD differed significantly between severity categories, averaging 45.85 ± 6.36 fL in the severe CAD group and 42.41 ± 3.25 fL in the mild-to-moderate group (p = 0.003). PDW values were similar between groups (15.60 ± 1.50 vs 15.80 ± 1.30 fL; p = 0.452). The remaining measured haematological and lipid variables showed no statistically significant group differences.

Table 2: Laboratory measurements by Gensini-defined CAD category

Laboratory measurement	Severe CAD (n = 29)	Mild-to-moderate CAD (n = 36)	p-value
Fasting blood glucose (mg/dL)	145.04 ± 50.15	126.47 ± 51.40	0.038[a]
Total cholesterol (mg/dL)	160.38 ± 5.41†	153.28 ± 39.00	0.524[b]
Triglycerides (mg/dL)	170.21 ± 90.64	152.11 ± 57.00	0.930[a]
LDL cholesterol (mg/dL)	69.49 ± 39.50	69.58 ± 33.26	0.746[a]
HDL cholesterol (mg/dL)	51.21 ± 26.00	44.50 ± 9.86	0.735[a]
VLDL cholesterol (mg/dL)	32.40 ± 17.60	29.30 ± 12.70	0.935[a]
White blood cell count (× 10^9/L)	8.71 ± 2.66	7.15 ± 2.11	0.005[a]
Platelet count (× 10^9/L)	234.72 ± 60.65	206.44 ± 49.04	0.424[b]
RDW-SD (fL)	45.85 ± 6.36	42.41 ± 3.25	0.003[a]
PDW (fL)	15.60 ± 1.50	15.80 ± 1.30	0.452[a]

Note: The total-cholesterol SD (5.41 mg/dL) is retained exactly as reported in the original study table because the raw dataset was not available for independent recalculation. Values are presented as mean ± standard deviation. CAD, coronary artery disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PDW, platelet distribution width; RDW-SD, red cell distribution width-standard deviation; VLDL, very-low-density lipoprotein; [a] Independent-samples t-test; [b] Mann-Whitney U test

Correlation Analysis

Across all participants, the Gensini score increased as RDW-SD increased, with a Spearman correlation coefficient of 0.358 (p = 0.003), indicating a moderate positive association. This relationship is illustrated in Figure 1.

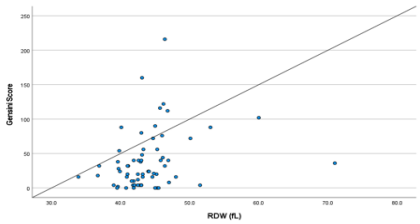


Figure 1. Scatterplot of RDW-SD and Gensini score
RDW-SD, red cell distribution width-standard deviation

Receiver Operating Characteristic Analysis
For identifying severe CAD, RDW-SD produced an AUC of 0.710 (95% CI, 0.582–0.837), consistent with limited-to-moderate discrimination. The corresponding AUC for PDW was approximately 0.43, indicating poor discriminatory performance. These results are presented in Figure 2.

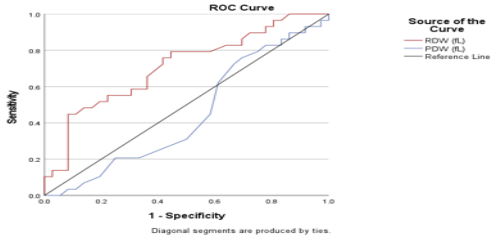


Figure 2. Receiver operating characteristic curves for RDW-SD and PDW
CAD, coronary artery disease; PDW, platelet distribution width; RDW-SD, red cell distribution width-standard deviation; ROC, receiver operating characteristic

Logistic Regression Analysis
In the adjusted logistic regression model, each one-unit increase in RDW-SD was associated with higher odds of severe CAD (adjusted OR, 1.320; 95% CI, 1.055–1.651; p = 0.015). No other included covariate reached statistical significance in the adjusted model. These findings are presented in Table 3. The number of observations included in the fitted model was 65. Given the small number of severe CAD events relative to the number of covariates, these adjusted estimates should be interpreted as exploratory and potentially unstable.

Table 3: Multivariable logistic regression analysis of factors associated with severe coronary artery disease

Variable	p-value	Adjusted OR	95% CI
Hypertension	0.361	0.560	0.161–1.946
Diabetes mellitus	0.102	2.847	0.812–9.985
LDL cholesterol (mg/dL)	0.449	1.007	0.990–1.024
White blood cell count (× 10^9/L)	0.381	1.153	0.839–1.585
Platelet count (× 10^9/L)	0.259	1.008	0.994–1.021
RDW-SD (fL)	0.015	1.320	1.055–1.651
PDW (fL)	0.429	0.527	—

Note: The PDW 95% confidence interval is not reported because the interval in the source table (0.527–1.312 for OR 0.527) is internally inconsistent. The reported OR and p-value are retained without recalculation.
Dependent variable: Severe CAD (Gensini score ≥ 32); Variables included in the model: hypertension, diabetes mellitus, LDL cholesterol, white blood cell count, platelet count, RDW-SD and PDW; CAD, coronary artery disease; CI, confidence interval; LDL, low-density lipoprotein; OR, odds ratio; PDW, platelet distribution width; RDW-SD, red cell distribution width-standard deviation

Discussion

This study examined whether two indices obtained from a routine full blood count reflect angiographic CAD extent in patients with stable angina. The principal finding was that RDW-SD was higher in patients above the severe CAD threshold, increased in parallel with the continuous Gensini score, and retained a statistically significant association in the adjusted model. PDW, by contrast, did not vary meaningfully with disease extent in this cohort. These findings distinguish a potentially informative erythrocyte index from a platelet index that offered little discriminatory value in this sample.

The observation that men were overrepresented in the severe CAD category is consistent with established epidemiological patterns, although interpretation requires caution given the small sample size and the absence of information on menopausal status and other sex-specific factors (14, 15). Smoking and diabetes mellitus were also more frequent in the severe CAD group, but neither comparison met the prespecified significance threshold, and diabetes was not significant after adjustment. These patterns are compatible with the known contribution of conventional risk factors to coronary atherosclerosis but cannot confirm group-specific effects in this dataset.

Severe disease was accompanied by higher fasting glucose and white blood cell count. Altered glucose regulation and systemic inflammation could plausibly underlie these observations, but the cross-sectional design cannot establish mechanism or temporal direction. The leukocyte association also disappeared in the adjusted analysis, suggesting that it may be explained by other variables in the model or may reflect limited statistical power.

The prevalence of severe CAD in this cohort (44.6%) is broadly consistent with findings from a Nigerian tertiary centre, where patients undergoing angiography for suspected CAD frequently presented with advanced disease[4]. Among the two cell-distribution measures, RDW-SD demonstrated the more consistent relationship with coronary burden. Greater red cell size dispersion may arise from inflammatory or oxidative stress, nutritional deficits or impaired erythrocyte production (6, 7). The observed correlation supports an association with more extensive atherosclerosis and accords with previous reports linking increased RDW to cardiovascular risk (8, 9, 16). However, the present study does not identify the biological pathway responsible, and the possibility of residual confounding by unmeasured factors such as anaemia, iron status, folate or vitamin B12 deficiency, renal function or medication exposure cannot be excluded.

RDW-SD classified severe CAD only moderately, with an AUC of 0.710. The confidence interval around this estimate was broad, extending from 0.582 to 0.837, and is compatible with considerably weaker discrimination than the point estimate suggests. Routine availability and negligible additional cost make RDW-SD attractive as a potential supplementary measure, but the present findings do not demonstrate that it improves risk prediction beyond established clinical assessment. Any practical role would require demonstration of incremental value in a prospectively validated setting.

There was no convincing PDW–CAD correlation when used with categorical CAD severity; a PDW–Gensini correlation coefficient was not provided within the provided materials. Antiplatelet exposure and pre-analytical procedures and sampling differences could also have dampened any relationship in this relatively limited group of patients in the stable angina group.12 The lack of a statistically significant association does not imply that PDW is of no clinical value, but rather that there is no compelling evidence for clinical use of PDW in this context.

Study Limitations

Several constraints limit the interpretation of this study. Recruitment from a single centre and the modest sample size reduce generalisability to other populations and settings. The cross-sectional design prevents causal inference and cannot establish whether elevated RDW-SD precedes or follows the development of severe coronary atherosclerosis. The severe CAD group contained only 29 patients relative to the number of regression covariates, so adjusted estimates may be unstable and should be regarded as exploratory. The Gensini threshold of 32, although prespecified, represents a dichotomisation of a continuous variable and may not capture the full spectrum of disease severity. Haemoglobin-related indices, iron, folate, vitamin B12, inflammatory biomarkers and detailed treatment exposure were not fully evaluated, and the absence of these variables may contribute to residual confounding. Laboratory measurements were performed using institutional procedures that were not fully documented, limiting reproducibility. Longitudinal changes in RDW-SD and subsequent clinical events were not available, and the ROC analysis was exploratory without external validation. The possibility of chance findings arising from multiple comparisons should also be considered.

Future Directions

Prospective multicentre studies with larger sample sizes are needed to confirm the present findings and to determine whether RDW-SD provides clinically useful information beyond established cardiovascular risk factors. Such studies should use prespecified severity thresholds, standardised laboratory measurements with documented quality control, a justified sample size and clinically justified adjustment for confounders. Independent validation of any proposed diagnostic threshold is essential before clinical application is considered. If clinical utility is the ultimate objective, future investigations should also evaluate whether RDW-SD adds discrimination

and calibration beyond conventional predictors, and whether it improves risk stratification in ways that alter clinical decision-making.

Conclusion

In this single-centre cross-sectional study of patients investigated for stable angina, RDW-SD was positively associated with Gensini-defined angiographic CAD severity, whereas PDW showed no clear association. RDW-SD demonstrated limited-to-moderate discrimination for severe CAD. These findings are preliminary and are limited by the sample size, potential instability of the adjusted model and absence of external validation. Larger studies using prespecified analyses are needed to determine whether RDW-SD provides clinically useful information beyond established cardiovascular risk factors.

List of Abbreviations

AUC: Area under the curve
CAD: Coronary artery disease
CI: Confidence interval
HDL: High-density lipoprotein
LAD: Left anterior descending artery
LCX: Left circumflex artery
LDL: Low-density lipoprotein
OR: Odds ratio
PDW: Platelet distribution width
RDW-SD: Red cell distribution width-standard deviation
ROC: Receiver operating characteristic
VLDL: Very-low-density lipoprotein
WBC: White blood cell

Declarations

Ethics Approval and Consent to Participate

The study protocol was approved by the Scientific and Ethics Committee of Mosul Cardiac Centre (Approval No. 11; 18 November 2024). This work was carried out in compliance with the Declaration of Helsinki. All participants gave written informed consent before entering the study, and confidentiality was ensured throughout the study.

Consent for Publication

All authors gave consent for publication of the work under the Creative Commons Attribution Non-Commercial 4.0 licence.

Data Availability

The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare no competing interests.

Funding Sources

The work was undertaken without external financial support.

Author Contributions

HAM worked on conceptualisation, methodology, investigation, formal analysis, writing the original draft, writing the review and editing, and approval of the final manuscript.

The authors should make sure that the contributions of the other listed authors are correct before submission to report the CRediT properly.

Acknowledgements

The author acknowledges the clinical and laboratory personnel of Mosul Cardiac Centre who supported participant assessment and data collection.

References

1. World Health Organization. Cardiovascular diseases (CVDs) fact sheet. Geneva: WHO; 2021. Available from: https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds)
2. Yusuf S, Rangarajan S, Teo K, Islam S, Li W, Liu L, et al. Cardiovascular risk and events in 17 low- middle- and high-income countries. N Engl J Med. 2014;371(9):818-27. https://doi.org/10.1056/NEJMoa1311890
3. Amen SO, Baban ST, Yousif SH, Hawez AH, Baban ZT, Jalal DMF. Prevalence of the most frequent risk factors in Iraqi patients with acute myocardial infarction. Med J Babylon. 2020;17(1):6-18. https://doi.org/10.4103/MJBL.MJBL_66_19
4. Ojo O, Oguntola BO, Odele RO, Nnaji FC, Ojogbane F. Clinical characteristics, risk factors and angiographic profile of patients with suspected coronary artery disease undergoing coronary angiography in Ekiti, South-West, Nigeria. Babcock Univ Med J. 2025;8(1):46-52. https://doi.org/10.38029/babcockuniv.med.j..v8i1.597
5. Neumann FJ, Sechtem U, Banning AP, Bonaros N, Bueno H, Bugiardini R, et al. 2019 ESC Guidelines for the diagnosis and management of chronic coronary syndromes. Eur Heart J. 2020;41(3):407-77. https://doi.org/10.1093/eurheartj/ehz425
6. Gensini GG. A more meaningful scoring system for determining the severity of coronary heart disease. Am J Cardiol. 1983;51(3):606. https://doi.org/10.1016/S0002-9149(83)80105-2
7. Semba RD, Patel KV, Ferrucci L, Sun K, Roy CN, Guralnik JM, et al. Serum antioxidants

and inflammation predict red cell distribution width in older women: the Women's Health and Aging Study I. Clin Nutr. 2010;29(5):600-4. https://doi.org/10.1016/j.clnu.2010.03.001
8. Lippi G, Targher G, Montagnana M, Salvagno GL, Zoppini G, Guidi GC. Relation between red blood cell distribution width and inflammatory biomarkers in a large cohort of unselected outpatients. Arch Pathol Lab Med. 2009;133(4):628-32. https://doi.org/10.5858/133.4.628
9. Tonelli M, Sacks F, Arnold M, Moye L, Davis B, Pfeffer M. Relation between red blood cell distribution width and cardiovascular event rate in people with coronary disease. Circulation. 2008;117(2):163-8. https://doi.org/10.1161/CIRCULATIONAHA.107.727545
10. Uyarel H, Ergelen M, Cicek G, Kaya MG, Ayhan E, Turkkan C, et al. Red cell distribution width as a novel prognostic marker in patients undergoing primary angioplasty for acute myocardial infarction. Coron Artery Dis. 2011;22(3):138-44. https://doi.org/10.1097/MCA.0b013e328342c77b
11. Vagdatli E, Gounari E, Lazaridou E, Katsibourlia E, Tsikopoulou F, Labrianou I. Platelet distribution width: a simple, practical and specific marker of activation of coagulation. Hippokratia. 2010;14(1):28-32.
12. Sato Y, Yoshihisa A, Watanabe K, Yokokawa T, Misaka T, Kanno Y, et al. Association between platelet distribution width and prognosis in patients with heart failure. PLoS One. 2020;15(12):e0244608. https://doi.org/10.1371/journal.pone.0244608
13. Terakura M, Sugawara T, Hirota D, Sagawa T, Sakamoto T. Red cell and platelet distribution widths in patients with angina pectoris and acute myocardial infarction. Acute Med Surg. 2016;3(3):244-9. https://doi.org/10.1002/ams2.187
14. Khot UN, Khot MB, Bajzer CT, Sapp SK, Ohman EM, Brener SJ, et al. Prevalence of conventional risk factors in patients with coronary heart disease. JAMA. 2003;290(7):898-904. https://doi.org/10.1001/jama.290.7.898 https://doi.org/10.1001/jama.290.7.898
15. Maas AHEM, Appelman YEA. Gender differences in coronary heart disease. Neth Heart J. 2010;18(12):598-603. https://doi.org/10.1007/s12471-010-0841-y
16. Yusuf PS, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937-52. https://doi.org/10.1016/S0140-6736(04)17018-9