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Relationship Between Platelet Distribution Width and the Number of Coronary Artery Lesions in Patients with Acute Coronary Syndrome

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ABSTRACT

Introduction: Acute coronary syndrome (ACS) results from atherosclerotic plaque and thrombosis, with inflammation and platelet activation involved. Platelet distribution width (PDW), a marker of platelet activation, may reflect involvement. Study evaluated association with diseased vessels in ACS.

Methods: This cross-sectional study with retrospective data collection analyzed 111 patients admitted with ACS to H. Adam Malik General Hospital, Medan, between January and December 2024. Demographic, laboratory, and coronary angiographic data were retrieved from medical records. Patients were classified as having single-vessel or multi-vessel disease. Comparisons used the chi-square or Fisher exact test, the independent t-test, and the Mann–Whitney U test as appropriate; a receiver operating characteristic (ROC) curve was constructed to derive the optimal PDW cut-off. Analyses were performed in SPSS version 29, with significance set at $p < 0.05$.

Results: Of the 111 patients, 47 (42.3%) had single-vessel disease and 64 (57.7%) had multi-vessel disease. PDW was significantly higher in the multi-vessel disease group (median 11.0 vs. 10.7 fL; $p = 0.002$). ROC analysis yielded an area under the curve of 0.68 (95% CI 0.57–0.78) at a PDW cut-off of 10.25 fL, with a sensitivity of 95% and a specificity of 43%. A PDW ≥ 10.25 fL was significantly associated with multi-vessel involvement ($p = 0.001$).

Conclusion: PDW was associated with the number of diseased coronary vessels in patients with ACS. As an inexpensive routine blood count parameter, PDW may serve as an adjunctive marker of coronary lesion complexity; however, modest specificity limits its use as a stand-alone diagnostic tool.

Keywords

Acute Coronary Syndrome, Coronary Lesion, Platelet Activation, Platelet Distribution Width, Thrombosis

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INTRODUCTION

Acute coronary syndrome (ACS) is an acute clinical condition characterized by myocardial ischemia resulting from obstruction of the coronary arteries by atherosclerosis and thrombosis. Inflammation and platelet activation are central to the pathogenesis of ACS, contributing to the formation and progression of atherosclerotic plaques and the complexity of coronary lesions. In the United States, ACS is diagnosed in approximately 15.5 million people, and the American Heart Association estimates that a myocardial infarction occurs approximately every 41 seconds, making coronary disease the leading cause of death in that country [1]. Platelets are pivotal in thrombogenesis and atherogenesis: platelet adhesion and aggregation drive

atherothrombotic events, and greater platelet activity confers a higher risk of atherothrombosis and worse outcomes following such events. Because platelet activity is linked to acute vascular disease, antiplatelet therapy carries a Class I recommendation for the treatment and secondary prevention of coronary artery disease, and platelet function has been associated with the risk of adverse events across several categories of coronary heart disease [2].

Platelet distribution width (PDW) is an index of platelet size heterogeneity. Elevated PDW values indicate a wide range of platelet volumes resulting from inflammation, platelet injury, and the presence of immature platelets [3]. Tzur et al. reported that higher PDW values are associated with advancing age, a range of cardiovascular disorders, renal dysfunction, diabetes mellitus, cerebrovascular disease, and malignancy, as well as statin and anticoagulant therapy; elevated PDW also correlates positively with mean platelet volume (MPV) and inversely with platelet count [4]. Several studies have examined the relationship between PDW and specific diseases, particularly coronary heart disease. Polat et al. stratified patients into low (≤ 11.8 fL) and high (> 11.8 fL) PDW groups and found that higher PDW was associated with more severe coronary disease, as reflected by a higher Global Registry of Acute Coronary Events (GRACE) score and a reduced Thrombolysis in Myocardial Infarction (TIMI) flow grade [5]. Bekler et al. reported that increased PDW (> 17%) was associated with a higher Gensini score [6]. Elmoniem et al. found that PDW could predict primary outcomes—cardiac arrest, recurrent myocardial infarction, stroke, arrhythmia, shock, and heart failure—as well as major adverse cardiac events (MACE), and that it correlated with measures of disease severity such as the SYNTAX, Gensini, and TIMI risk scores and ejection fraction; in that study a PDW cut-off of 48 yielded a sensitivity of 81% and a specificity of 66% for predicting MACE [7].

In Indonesia, Putri et al. described a significant correlation between PDW and troponin I concentrations in patients with acute myocardial infarction ($r^2 = 0.713$, $p < 0.001$), indicating that higher PDW values accompany higher troponin I levels. This correlation remained significant across the chest pain onset subgroups (< 6 hours: $r^2 = 0.647$, $p < 0.001$; > 6 hours: $r^2 = 0.756$, $p < 0.001$), suggesting a role for PDW in the rise of cardiac biomarkers during the early course of infarction [8]. Several reports have linked the presence of large platelets to adverse outcomes in ACS; increased megakaryocyte heterogeneity, driven by heightened thrombopoietic activity in the bone marrow, contributes to a higher peripheral PDW. Elevated PDW in patients with ACS may therefore reflect cytokine-driven thrombopoiesis [5]. However, few studies have specifically examined the relationship between PDW and the number of diseased coronary arteries in patients with ACS. Therefore, the present study aimed to analyze the association between PDW and the number of coronary artery lesions in patients with ACS.

METHOD

This was an analytical observational study employing a cross-sectional design with retrospective data collection. Secondary data were obtained from the medical records of patients treated in the inpatient wards of H. Adam Malik General Hospital, Medan. Data extraction was conducted from May to June 2025 and covered patients admitted between January and December 2024. The study population comprised patients who met the eligibility criteria. Patients were eligible for inclusion if they were admitted with a diagnosis of ACS at H. Adam Malik General Hospital and had complete medical records, complete blood count results, and coronary angiography findings. Patients were excluded if they had a history of HIV infection, malignancy, autoimmune disease, liver cirrhosis, or sepsis, or if they were receiving non-steroidal anti-inflammatory drugs, steroids, hormonal therapy, or immunomodulators. Based on coronary angiography, patients were classified as having single-vessel or multivessel disease.

All analyses were performed using SPSS version 29. Differences between the single- and multi-vessel groups were assessed according to the type and distribution of each variable. For categorical variables, the chi-square test was applied when assumptions were met, and the Fisher ’sexact test was used otherwise. For numerical variables compared between the two groups, the independent t-test was used for normally distributed data and the Mann–Whitney U test for non-normally distributed data. A receiver operating characteristic (ROC) curve was constructed to determine the optimal PDW cutoff value, area under the curve (AUC), and

corresponding sensitivity and specificity. A p-value < 0.05 was considered statistically significant. The study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Universitas Sumatera Utara, and H. Adam Malik General Hospital. Because only de-identified secondary data were analyzed, the requirement for individual informed consent was waived. All procedures were conducted in accordance with the principles of the Declaration of Helsinki.

RESULTS

A total of 111 patients met the eligibility criteria. Most patients had multi-vessel disease (64 patients, 57.7%), while single-vessel disease was present in 47 patients (42.3%). The demographic and clinical characteristics of the study participants are summarized in Table 1. Among these characteristics, only the type of ACS differed significantly between the groups (p = 0.001). Mean age was higher in the multi-vessel group. Multi-vessel disease was more prevalent among male patients, whereas single-vessel disease was more common among female patients. Across the normal-weight, overweight, and obese Grade I categories, multi-vessel disease was the more frequent pattern. Patients with a history of smoking, type 2 diabetes mellitus, hypertension, or dyslipidemia were also more commonly found in the multi-vessel disease group. By ACS subtype, unstable angina pectoris was associated mainly with single-vessel disease, whereas NSTEMI and STEMI were associated predominantly with multi-vessel disease.

Table 1. Demographic and clinical characteristics of the study participants

Variable	Single-vessel (n = 47)	Multi-vessel (n = 64)	p-value
Age (years), mean ± SD	56.7 ± 9.9	58.6 ± 8.4	0.297[a]
Sex, n (%)			0.143[b]
Male	30 (27.0)	49 (44.1)	
Female	17 (15.3)	15 (13.5)	
Body mass index, n (%)			0.563[b]
Underweight	1 (0.9)	0 (0)	
Normal weight	13 (11.7)	17 (15.3)	
Overweight	13 (11.7)	19 (17.1)	
Obesity grade I	10 (9.0)	19 (17.1)	
Obesity grade II	10 (9.0)	9 (8.1)	
Smoking status, n (%)			0.344[b]
Never smoked	19 (17.1)	20 (18.0)	
Former smoker	10 (9.0)	22 (19.8)	
Active smoker	18 (16.2)	22 (19.8)	
Type 2 diabetes mellitus, n (%)			0.270[b]
Non-diabetic	32 (28.8)	37 (33.3)	
Diabetic	15 (13.5)	27 (24.3)	
Hypertension, n (%)			0.342[b]
Non-hypertensive	18 (16.2)	19 (17.1)	
Hypertensive	29 (26.1)	45 (40.5)	
Type of ACS, n (%)			0.001[b]
UAP	17 (15.3)	10 (9.0)	
NSTEMI	5 (4.5)	25 (22.5)	
STEMI	25 (22.5)	29 (26.1)	
Dyslipidaemia, n (%)			0.816[b]
Non-dyslipidaemic	13 (11.7)	19 (17.1)	
Dyslipidaemic	34 (30.6)	45 (40.5)	

Note: ACS, acute coronary syndrome; BMI, body mass index; NSTEMI, non-ST-elevation myocardial infarction; SD, standard deviation; STEMI, ST-elevation myocardial infarction; UAP, unstable angina pectoris. Percentages are calculated relative to the total cohort (n = 111). [a] Independent t-test; [b] chi-squared test. Statistical significance is indicated in bold (p < 0.05).

The laboratory characteristics of the study participants are presented in Table 2. Of the laboratory parameters examined, only PDW differed significantly between the two groups (p = 0.002). The mean hemoglobin level was marginally higher in the multi-vessel group, whereas the mean platelet count was higher in the single-vessel group. The mean random blood glucose level was higher among patients with multi-vessel disease, and the mean creatinine level was comparable between groups, whereas the median estimated

glomerular filtration rate (eGFR) was slightly lower in the multi-vessel group. Within the lipid profile, median total cholesterol and triglycerides and mean HDL cholesterol were higher in the single-vessel group, whereas mean LDL cholesterol was higher in the multi-vessel group. PDW was significantly higher in patients with multi-vessel disease.

Table 2. Laboratory characteristics of the study participants

Variable	Single-vessel (n = 47)	Multi-vessel (n = 64)	p-value
Haemoglobin (g/dL), mean ± SD	13.4 ± 2.6	13.6 ± 1.6	0.793[a]
Platelet count (×10³/μL), mean ± SD	268.1 ± 111.3	240.9 ± 60.0	0.088[a]
Random blood glucose (mg/dL), mean ± SD	168.4 ± 80.1	182.9 ± 95.6	0.380[a]
Creatinine (mg/dL), median (min–max)	1.1 (0.6–4.0)	1.2 (0.7–3.8)	0.339[b]
eGFR (mL/min/1.73 m²), median (min–max)	100 (14–100)	100 (18–100)	0.215[b]
Total cholesterol (mg/dL), median (min–max)	173.0 (85–571)	172.5 (108–270)	0.964[b]
Triglycerides (mg/dL), median (min–max)	160.0 (60–1026)	123.5 (67–595)	0.009[b]
HDL cholesterol (mg/dL), mean ± SD	41.2 ± 10.4	40.5 ± 9.8	0.788[a]
LDL cholesterol (mg/dL), mean ± SD	118.7 ± 35.0	122.4 ± 38.0	0.340[a]
PDW (fL), median (min–max)	10.7 (7.8–15.1)	11.0 (10.2–19.5)	0.002[b]

Note: eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PDW, platelet distribution width; SD, standard deviation. Independent t-test; Mann–Whitney U test. Statistical significance is indicated in bold ($p < 0.05$).

In the ROC and AUC analyses, PDW demonstrated modest discriminatory ability for the number of coronary lesions in ACS (AUC = 0.68; 95% CI 0.57–0.78); this association reached statistical significance (p = 0.002) (Figure 1, Table 3). The corresponding diagnostic performance showed high sensitivity (95%) but a relatively low specificity (43%), indicating that PDW identifies most patients with multi-vessel disease but is limited in excluding it.

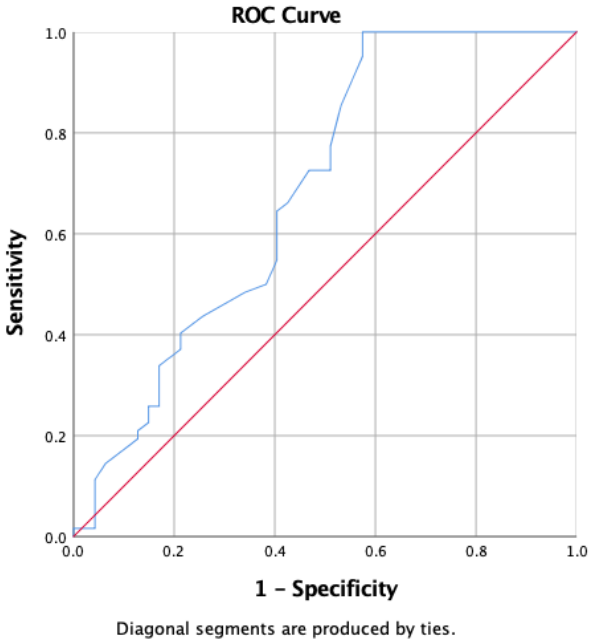

Figure 1. Receiver operating characteristic (ROC) curve for platelet distribution width in predicting the number of coronary artery lesions in acute coronary syndrome (AUC = 0.68).

Table 3. Diagnostic performance of PDW as a predictor of coronary lesion number in ACS

Variable	AUC (95% CI)	Cut-off (fL)	Sensitivity	Specificity	p-value
PDW	0.68 (0.57–0.78)	10.25	95%	43%	0.002

AUC, area under the curve; CI, confidence interval; PDW, platelet distribution width.

Using the derived cut-off of 10.25 fL, 88 patients (79.3%) had a PDW ≥ 10.25 fL, and 23 patients (20.7%) had a PDW < 10.25 fL (Table 4). Among patients with a PDW ≥ 10.25 fL, the majority (61 patients,

55.0% of the cohort) had multi-vessel disease, whereas patients with a PDW < 10.25 fL predominantly had single-vessel disease. Bivariate analysis confirmed a significant association between the PDW category and the number of coronary lesions in ACS (p = 0.001).

Table 4. PDW category and number of coronary artery lesions in ACS.

PDW (fL)	Total, n (%)	Single-vessel, n (%)	Multi-vessel, n (%)	p-value
< 10.25	23 (20.7)	20 (18.0)	3 (2.7)	0.001
≥ 10.25	88 (79.3)	27 (24.3)	61 (55.0)	

Note: PDW, platelet distribution width. Percentages are calculated relative to the total cohort (n = 111). Chi-square test; a p-value < 0.05 was considered significant (shown in bold).

DISCUSSION

This study evaluated whether PDW is associated with the number of diseased coronary vessels in patients with ACS at H. Adam Malik General Hospital, Medan. Among the 111 patients analyzed, the mean age was 56.7 ± 9.9 years in the single-vessel group and 58.6 ± 8.4 years in the multi-vessel group (p = 0.297); older age tended to occur in the multi-vessel group. This pattern is consistent with the findings of Siregar et al. and Du et al., who reported a higher prevalence of multi-vessel disease with advancing age [9,10]. Aging accelerates atherosclerosis through inflammatory mechanisms, particularly the interleukin-6 pathway, which drives the progression of coronary lesions [11]. Although not statistically significant here, this observation supports the view that aging is not merely a traditional risk factor but also a biological process that accelerates vascular injury and worsens disease severity, including the number of involved vessels.

Men predominated in both groups (single-vessel: 30 men [27.0%] and 17 women [15.3%]; multi-vessel: 49 men [44.1%] and 15 women [13.5%]; p = 0.143). This is in accordance with the findings of Mappangara et al., who observed a male predominance in ACS that was not consistently related to lesion number [12]. Estrogen is thought to protect the vascular endothelium and modulate lipid metabolism, thereby reducing the risk of atherosclerosis in premenopausal women, whereas men typically have greater exposure to risk factors, such as smoking, stress, and dyslipidemia. The male predominance in ACS, including multivessel disease, therefore, likely reflects both biological and behavioral contributions. With respect to body mass index, being overweight or having obesity was more frequent in the multivessel group (p = 0.563), in keeping with the findings of Mappangara et al., who found no significant association between obesity and lesion number [12]. This suggests that the metabolic and inflammatory milieu accompanying obesity may contribute more to the initiation of atherosclerosis than to its progression in terms of the number of lesions [13]. A history of smoking was more common in the multivessel group, although the difference was not significant (p = 0.344), consistent with [14]. Smoking promotes atherosclerosis through oxidative stress, endothelial dysfunction, and inflammatory activation that accelerate plaque formation [15]. The lack of significance here probably reflects the multifactorial nature of coronary disease. Type 2 diabetes mellitus was likewise more frequent in the multivessel group (p = 0.270), in line with [9]. Hyperglycemia and insulin resistance accelerate atherosclerosis by injuring the vascular endothelium and promoting atheroma formation, particularly under hyperglycemic stress [16]. Despite the absence of statistical significance, diabetes remains a key biological contributor to lesion complexity. Hypertension was also more prevalent in the multivessel group (p = 0.342), again in accordance with Siregar et al., although other studies have reported conflicting results [12].

Hypertension is a major cardiovascular risk factor that promotes the development of more complex vascular lesions through endothelial injury, chronic inflammation, and hemodynamic stress. Sustained elevation of blood pressure induces arterial wall stress, endothelial dysfunction, increased vascular permeability, and activation of the renin–angiotensin–aldosterone system, leading to vascular remodelling and complex plaque formation; it also amplifies the effects of hyperglycemia and dyslipidemia [16]. According to the ACS subtype, the single-vessel group comprised 17 patients with UAP (15.3%), five with NSTEMI (4.5%), and 25 with STEMI (22.5%), whereas the multi-vessel group comprised 10 with UAP (9.0%), 25 with NSTEMI (22.5%), and 29 with STEMI (26.1%) (p = 0.001). This significant difference—with NSTEMI and STEMI occurring more frequently in multi-vessel disease—mirrors Siregar et al., who reported that most multi-vessel patients had STEMI (57.1%), followed by NSTEMI (32.7%) and unstable angina (10.2%), with

ACS subtype the only variable showing a significant difference [9]. Pathophysiologically, multi-vessel involvement increases the ischemic burden and risk of plaque instability, often manifesting as myocardial infarction rather than the milder or transient stenosis underlying UAP [17]. These findings reinforce the link between coronary lesion complexity and ACS severity and the need for more aggressive revascularization and risk-management strategies in multi-vessel disease.

Dyslipidemia was more common in the multi-vessel group (p = 0.816), consistent with a previous study [12]. Dyslipidemia remains a major risk factor for coronary artery disease: elevated LDL cholesterol promotes lipid accumulation in the arterial wall and plaque formation, whereas reduced HDL cholesterol impairs reverse cholesterol transport [16]. Thus, although not significant in this cohort, dyslipidemia remains an important contributor to lesion development, and optimal lipid control—lowering LDL and raising HDL—remains central to prevention. Among the haematological parameters, the mean hemoglobin level was 13.4 ± 2.6 g/dL in the single-vessel group and 13.6 ± 1.6 g/dL in the multi-vessel group (p = 0.793; not significant), in agreement with a previous report [9]. The mean platelet count was 268.1 ± 111.3 $\times 10^3/\mu L$ versus 240.9 ± 60.0 $\times 10^3/\mu L$ (p = 0.088), and the mean random blood glucose level was 168.4 ± 80.1 mg/dL versus 182.9 ± 95.6 mg/dL (p = 0.380); neither differed significantly, consistent with previous reports [12]. These parameters largely reflect short-term states rather than chronic atherosclerotic processes, although anemia may aggravate myocardial ischemia and elevated platelet counts may increase thrombotic risk [18]. The median creatinine level was 1.1 mg/dL (0.6–4.0) in the single-vessel group and 1.2 mg/dL (0.7–3.8) in the multi-vessel group (p = 0.339), with no significant difference, consistent with Siregar et al. The median eGFR was 100 (14–100) versus 100 (18–100) mL/min/1.73 m^2 (p = 0.215), slightly lower in the multi-vessel group, in line with He et al., who found lower eGFR to be significantly related to multi-vessel disease [16]. Declining renal function contributes to oxidative stress and chronic inflammation (raised CRP and IL-6) and vascular calcification through calcium–phosphate imbalance, thereby accelerating atherosclerosis and the development of multiple lesions [19]. Within the lipid profile, median total cholesterol was 173.0 mg/dL (85–571) versus 172.5 mg/dL (108–270) (p = 0.964; not significant) [12]. whereas median triglycerides were significantly higher in the single-vessel group (160.0 mg/dL [60–1026] vs. 123.5 mg/dL [67–595]; p = 0.009), consistent with [16]. This difference may reflect population variation, lipid-lowering therapy, or acute metabolic stress rather than chronic vascular disease, as triglycerides indicate atherogenic remnant lipoproteins, whose acute elevation can accompany the early phase of ACS.

The mean HDL-C level was 41.2 ± 10.4 mg/dL in the single-vessel group and 40.5 ± 9.8 mg/dL in the multi-vessel group (p = 0.788; not significant), in agreement with a previous study [12]. HDL protects against atherosclerosis by inhibiting monocyte adhesion and LDL oxidation, and low or dysfunctional HDL is associated with greater coronary risk [14]. The mean LDL-C level was 118.7 mg/dL versus 122.4 ± 38 mg/dL (p = 0.340; not significant), consistent with previous reports [14,16]. Although LDL elevation is theoretically linked to coronary risk, these findings suggest that serum HDL-C and LDL-C alone may not capture atherosclerotic activity during ACS owing to acute-phase lipid shifts, oxidative stress, and systemic inflammation. Notably, dyslipidemia was assessed categorically by history in Table 1 and quantitatively in Table 2, which may explain apparent discrepancies: higher triglycerides in single-vessel cases may reflect acute inflammatory lipolysis during early ACS, whereas the slightly higher LDL-C in multi-vessel cases may indicate chronic dyslipidemia driving plaque progression. Pre-admission statin use may further account for the inconsistent triglyceride and LDL-C differences, underscoring that lipid profiles in ACS should be interpreted in the context of clinical phase, metabolic control, and statin adherence [20]. Patients with multi-vessel disease generally carry high-risk comorbidities that contribute to poorer prognosis. Earlier work has shown that patients with simultaneous plaque rupture and acute multi-vessel percutaneous coronary intervention face higher mortality and reinfarction risk, and that multi-vessel disease independently predicts worse long-term outcomes, partly because of lower reperfusion success [21]. In the present study, the median PDW was 10.7 fL (7.8–15.1) in the single-vessel group and 11.0 fL (10.2–19.5) in the multi-vessel group (p = 0.002), being significantly higher in multi-vessel disease. This agrees with Mappangara et al., who found PDW to be significantly correlated with multi-vessel lesions [12]. Although De Luca et al. reported no significant

difference in PDW between single- and multi-vessel disease [22]. ROC analysis showed that PDW had modest discriminatory power (AUC = 0.68) for lesion number, with high sensitivity (95%) but low specificity (43%) at a cut-off of 10.25 fL. Approximately 79.3% of patients had a PDW ≥ 10.25 fL, and among these, the majority (55%) had multi-vessel disease, whereas those below the cut-off were predominantly single-vessel; bivariate analysis confirmed a significant association between PDW and lesion number.

reported a higher PDW cutoff of 15.55 fL (AUC = 0.640) for predicting coronary artery disease, with moderate discrimination and a sensitivity and specificity of 61.5% and 55.8%, respectively [23]. found PDW to be an independent predictor of no-reflow and in-hospital MACE in STEMI patients undergoing percutaneous coronary intervention, and a cutoff of 15.8% predicted MACE with 79% sensitivity and 47% specificity [24]. The cutoff in the present study (10.25 fL) is lower than these values, which may reflect differences in study populations; the inflammatory and platelet activation profile of an Indonesian ACS cohort may differ from that of Indian or Turkish cohorts. In addition, the higher proportion of multi-vessel patients (55%) may have shaped the ROC curve, increasing sensitivity and shifting the threshold downward. Therefore, the cutoff likely reflects local population characteristics and may be most relevant regionally [24]. reported significantly higher mean PDW in patients with ACS than in controls (18.2 ± 1.52 vs. 16.8 ± 1.28 fL; $p < 0.001$), with the highest values in STEMI, followed by NSTEMI and unstable angina [25]. By contrast, Turk et al. found no significant difference in PDW between the STEMI and NSTEMI/UAP groups and no correlation between the Gensini score and PDW [26]. similarly reported no significant association in selected subgroups [6]. These divergent findings highlight that, while PDW is a biologically plausible and accessible marker, its diagnostic thresholds and discriminatory value are influenced by population characteristics and clinical context. This study has several limitations. Its cross-sectional, single-center, retrospective design precludes inferences about causality and limits generalizability. Reliance on secondary data restricted control over potential confounders, such as pre-admission medication, the timing of blood sampling relative to symptom onset, and inter-assay variability in platelet indices. PDW was analyzed in isolation rather than alongside other platelet volume indices, such as MPV or the platelet–large cell ratio, and lesion burden was quantified by the number of diseased vessels rather than by validated angiographic severity scores, such as the SYNTAX or Gensini score. Prospective, multicenter studies incorporating standardized platelet index measurement, comprehensive angiographic scoring, and clinical outcome follow-up are warranted to confirm and extend these findings.

CONCLUSION

Platelet distribution width (PDW) was associated with the number of diseased coronary vessels in patients with ACS. Platelet count was higher in single-vessel disease, whereas random blood glucose was higher and eGFR lower in multi-vessel disease; LDL cholesterol was also higher in multi-vessel disease. PDW was higher in the multi-vessel group and, at a cutoff of 10.25 fL, showed high sensitivity but limited specificity for predicting lesion number. As an inexpensive routine complete blood count parameter, PDW may be an adjunctive marker of coronary lesion complexity in ACS and should complement established diagnostic and angiographic assessments.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The authors agree to the publication of this article in the Journal of Society Medicine.

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All authors have reviewed and approved the final version of the manuscript and have agreed to its publication in the Journal of Society Medicine.

AUTHORS' CONTRIBUTIONS

All authors have reviewed and approved the final version of the manuscript, and they all agree to be accountable for all aspects of the work.

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