Platelet Indices as a marker of severity in Acute Coronary Syndrome patients at Qena University Hospital

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Abstract

Background: Coronary artery disease (CAD) is the leading global cause of death, with acute coronary syndrome (ACS) being a severe form leading to myocardial injury. Platelets and lipids play a key role in thrombosis and atherogenesis. Complete blood count (CBC) ratios may predict ACS outcomes.

Objectives: To assess the relationship between platelet and inflammatory indices and the severity of ACS.

Patients and methods: This cross-sectional study involved ACS patients aged 18+. Patients were divided into groups 1 (stable CAD) and 2 (acute myocardial infarction). Clinical and laboratory assessments included ECG, echocardiography, CBC, cardiac enzymes, and inflammatory markers.

Results: Cases had significantly higher systolic blood pressure; ECG was abnormal in 95%. ST depression was present in 59% of patients, 53% had an ejection fraction (EF) of less than 50%; and the mean EF was 48.09±8.93%. Patients with EF<50% had higher platelet count (336.962±73.198 vs 246.83±54.308, P<0.0001) and increased inflammatory indices (P < 0.05), but lower mean platelet volume (MPV) (8.91±2.72 vs 11.92±3.57, P<0.0001) and lymphocyte count (2.551±1.211 vs 2.989±1.298, P=0.0297). Neutrophil platelets score (NPS) and prognostic index (PI) scores of 1 and 2 were more prevalent among ACS cases with EF of < 50% (P<0.05). Platelet distribution width (PDW) and systemic immune-inflammation index (SII) were independent positive predictors of left ventricular ejection fraction (LVEF) (P<0.0001), whereas C-reactive protein (CRP) level was a significant independent negative predictor (P<0.0001).

Conclusion: Hematologic inflammatory indices and EF were correlated in ACS patients; higher ratios are associated with systemic inflammation and reduced EF.

Keywords: Inflammatory and platelet indices; SII; LVEF; Neutrophil platelet score.

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Introduction

Coronary artery disease (CAD) is the leading cause of illness and death worldwide, and acute coronary syndrome (ACS) is a severe form of sudden decrease in blood flow, causing myocardial ischemia or infarction. ACS is classified as ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and unstable angina. Elevated levels of cardiac troponins (cTn I and T) are widely recognized as the preferred biomarkers for assessing myocardial injury **(Schiavone et al., 2020).**

Platelets are crucial in coronary thrombosis and atherogenesis, affecting the risk of cardiovascular disease (CVD) and lipid metabolism. Lipids activate platelets through inflammatory factors and peptide hormone receptors, which promote thrombosis **(Khodadi, 2020).**

Research suggests that combining complete blood count (CBC) parameters, such as the systemic immune-inflammation index (SII), neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR), may serve as valuable prognostic markers for ACS patients undergoing percutaneous coronary intervention (PCI) **(Fan et al., 2022).**

This study aimed evaluate the correlation between platelet indices and the severity of acute coronary syndrome.

Patients and methods

This cross-sectional study was conducted at Qena University Hospital, Egypt, and involved adult patients aged ≥18 years with an ACS diagnosis. Exclusion criteria included severe inflammatory or infectious diseases, connective tissue disorders, significant hepatic or renal impairment, myeloproliferative disorders, refusal to participate, younger age, marked anemia or thrombocytopenia, antiplatelet therapy or drugs stimulating bone marrow, autoimmune diseases, bleeding diathesis, major operative trauma, malignant hypertension, malignancy, pregnancy, and severe hepatic or renal impairment.

The sample comprised all patients diagnosed with ACS who attended the hospital between October 1, 2023, and September 30, 2024.

Patients were divided into two groups. Group 1 (control) included patients with stable coronary artery disease (SCAD) who presented with acute chest pain with no signs of myocardial ischemia on electrocardiograms (ECG), troponin I tests, or echocardiography (ECHO). Group 2 included patients with acute myocardial infarction (AMI) who presented with typical acute chest pain with ischemic changes and elevated troponin I levels.

This study involved comprehensive clinical and laboratory assessments on admission, including detailed medical history, recording baseline heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean blood pressure (MBP), and body mass index (BMI). An electrocardiogram (ECG) was performed to identify signs of myocardial ischemia, focusing on ischemic changes such as alterations in the ST segment, T wave, and Q wave. Echocardiography was performed using a Vivid 5S machine (General Electric) to assess wall motion abnormalities and to measure the left ventricular ejection fraction (LVEF), left ventricular end-diastolic diameter (LVEDD), and ejection fraction (EF).

Laboratory investigations involved the collection of 5 mL of venous blood from each patient under aseptic conditions. One milliliter was placed into EDTA tubes for complete blood count (CBC) analysis and 4 mL in plain tubes for biochemical tests. The CELL-DYN Ruby automated cell counter (Abbott Diagnostics, Santa Clara, California, USA) processed the CBC within

60 minutes, providing various parameters including platelet count (140–450 × 10^9/L) and indices such as mean platelet volume (MPV, 8–15 fl), platelet distribution width (PDW, 10–17%), platelet-large cell ratio (PLCR, 13–43%), and plateletcrit (PCT, 0.11–0.28%). Several platelet indices were calculated, including MPV/PCT, PDW/platelet count, and MPV/platelet count.

Cardiac enzyme levels were used to assess myocardial injury, including creatinine kinase-MB (CK-MB) (N: 5-25 U/L), troponin I (N: 0- 0.04 ng/mL), and lipid profile using a Beckman Coulter automated analyzer. If the initial measurements were negative, serial tests were performed 3-6 hours after symptom onset. Additionally, albumin (N: 3.5- 5.5 g/dL) was measured using the Architect Plus c4000 (Abbott, US). C-reactive protein (CRP) was determined using a latex-enhanced immunoturbidimetric assay (Beckman Coulter AU 480, CA, USA) (0-22 mg/dL).

The study analyzed several inflammatory indices and prognostic markers, including platelet-to-lymphocyte ratio (PLR) **(Hudzik et al., 2015; Gong et al., 2021),** platelet-to-neutrophil ratio (PNR) **(Jin et al., 2019),** neutrophil platelet score (NPS) **(Watt et al., 2015),** red cell distribution width-to-platelet count ratio (RDW/ platelets), neutrophil-to-lymphocyte ratio (NLR) **(Chen et al., 2020; Gong et al., 2021),** lymphocyte-to-monocyte ratio (LMR) **(Ren et al., 2017; Gong et al., 2021),** P2/MS ratio **(Loganathan, 2018),** systemic immune-inflammation index (SII) **(Huang, 2023; Xu et al., 2023),** platelet-to-NLR ratio (P/NLR) **(Milenkovic et al., 2022),** platelet-to-CRP ratio (P/CRP) **(Li et al., 2021),** WBC count-to-MPV ratio (WMR) **(Dehghani et al., 2016; Guet et al., 2019),** CRP-to-serum albumin ratio (CAR), and the neutrophil percentage-to-albumin ratio **Sun et al. (2020).**

Ethical approval code: SVU-MED-CCP031-1-23-3-592.

Statistical analysis

Statistical data were analyzed using SPSS 24. The Kolmogorov-Smirnov test was used to test data normality; data are presented as mean values ± standard deviation for normally distributed data and medians with interquartile ranges for non-normally distributed data. Categorical variables are expressed as frequency and percentage. Student's t-test or Mann-Whitney test for quantitative data, one-way ANOVA for comparing means across more than two groups, and Chi-square or Fisher's exact tests for non-parametric data. Correlations were tested using the Pearson' test. Binary stepwise logistic regression revealed independent ACS risk variables, including univariate predictors in the multivariate model. Since p-values were < 0.05, adjusted odds ratios with 95% confidence intervals were determined.

Results

The study found no significant age difference between cases and controls, with males comprising 56% of cases and 56.67% of controls. A positive family history of CAD was present in 11% of cases. ACS cases had higher mean systolic blood pressure than controls (127.36 ± 11.82 vs. 111.07 ± 0.77 mmHg, P < 0.0001), lower weight (P = 0.0004) and height (P = 0.0001), but similar BMI. ECG was abnormal in 95% (P < 0.0001), and ST depression was present in 59% (P = 0.0003). The LVEF was significantly lower in the cases (48.09 ± 8.93% vs. 61.57 ± 4.18%), and 53% were < 50%. (**Table.1).**

Table 1. Basal characteristics of the studied population

Variables	ACS Cases (AMI) (N = 100)	(Stable CAD) Control (N= 30)	P-value
Age (Years), Mean ± SD	50.41 ± 11.64	46.23 ± 9.83	0.0593 [s.t.]
Sex, N (%)			
• Male	56 (56%)	17 (56.67%)	0.949 [χ2]
• Female	44 (44%)	13 (43.33%)	
Family history of CAD, N (%)			
• Positive	11 (11%)	0 (0%)	0.0672 [f]
• Negative	89 (89%)	30 (100%)	
Smoking, N (%)			
• Non-smoker	68 (68%)	15 (50%)	0.0002* [χ^2]
• Smoker	26 (26%)	6 (20%)	
• Ex-smoker	6 (6%)	9 (30%)	
Alcohol drinking, N (%)			
• Alcoholic	2 (2%)	0 (0%)	0.99 [f]
• Non-alcoholic	98 (98%)	30 (100%)	
Comorbidities, N (%)			
• Diabetes mellitus	29 (29%)	4 (13.33%)	0.085 [χ^2]
• Hypertension	36 (36%)	6 (20%)	0.136 [χ^2]
• Hyperlipidemia	30 (30%)	0 (0%)	0.059 [f]
• Atrial fibrillation	13 (13%)	0 (0%)	0.6038 [f]
• History of stroke	9 (9%)	0 (0%)	0.99 [f]
Blood pressure			
• SBP (mmHg)	127.36 ± 11.82	111.07 ± 0.77	<0.0001* [MWU]
• DBP (mmHg)	78.34 ± 8.26	76.1 ± 5.2	0.2569 [MWU]
Heart rate (beat/min)	82.81 ± 7.7	82.23 ± 12.35	0.8877 [MWU]
Anthropometrics			
• Weight (Kg)	71.96 ± 8.88	77.6 ± 6.96	0.0004* [MWU]
• Height (cm)	165.22 ± 5.95	170.93 ± 6.49	0.0001* [MWU]
BMI (Kg/m^2)	26.5 ± 4	26.68 ± 3.26	0.5636 [MWU]
ECG, N (%)			
• Normal ECG	5 (5%)	30 (100%)	<0.0001* [χ2]
• ST elevation	31 (31%)	0 (0%)	0.0593 [f]
• ST depression	59 (59%)	0 (0%)	0.0003* [f]
• Inverted T Wave	5 (5%)	0 (0%)	0.99 [f]
Ejection fraction (%)	48.09 ± 8.93	61.57 ± 4.18	<0.0001* [MWU]
• < 50%	53 (53%)	0 (0%)	<0.0001* [f]
• ≥ 50%	47 (47%)	30 (100%)	

*: significant; s.t.: student t-test; MWU: Mann Whitney U-test; f: Fisher exact test; χ2: Chi-square test; CAD: coronary artery disease; SBP: systolic blood pressure; DBP: diastolic blood pressure; ECG: electrocardiogram; BMI: body mass index.

Patients with ACS had higher mean Hb levels ($P < 0.0001$), platelet count ($P < 0.0001$), PCT ($P = 0.0007$), MPV ($P = 0.0028$), PDW ($P < 0.0001$), WBC ($P < 0.0001$), neutrophil count ($P = 0.0004$), and lymphocyte count ($P = 0.0004$). ACS cases had higher mean CRP levels (104.56 ± 87.68 vs. 3.53 ± 1.8 mg/L, $P < 0.0001$); troponin I and CK-MB were significantly higher. The inflammatory indices PMI, WBCs/MPV ratio, PLR, SII, and P2/MS were higher in ACS cases, with more cases having a PI score of 1 and 2 than the control ($P < 0.0001$). Serum albumin levels ($P = 0.0024$), RDW/platelet ratio, and lymphocyte/CRP, albumin/CRP, and platelet/CRP ratios were lower (all $P < 0.05$). (**Table.2).**

Table 2. CBC, biochemical markers, and inflammatory indices in the studied group

Variables	ACS Cases (AMI) (N = 100)	(Stable CAD) Control (N = 30)	P-Value
CBC parameters			
Hemoglobin (g/dl)	13.91 ± 1.63	13.04 ± 0.64	<0.0001* [MWU]
Platelet count (×103/µL)	294.6 ± 79.054	231.967 ± 24.314	<0.0001* [MWU]
Mean Platelet Volume (MPV fL)	10.33 ± 3.49	8.36 ± 1.01	0.0028* [MWU]
Plateletcrit (PCT %)	0.28 ± 0.04	0.24 ± 0.09	0.0007* [MWU]
Platelet distribution width (PDW %)	3.98 ± 1.13	2.71 ± 0.45	<0.0001* [MWU]
White blood cells WBCs ($\times 10^3/\mu L$)	11.456 ± 3.46	8.167 ± 1.949	<0.0001* [MWU]
Monocyte count ($\times 10^3/\mu L$)	0.506 ± 0.217	0.416 ± 0.176	0.0694 [MWU]
Neutrophil count ($\times 10^3/\mu L$)	5.851 ± 1.91	4.634 ± 1.164	0.0004* [MWU]
Eosinophil count ($\times 10^3/\mu L$)	0.232 ± 0.176	0.212 ± 0.116	0.7337 [MWU]
Lymphocyte count ($\times 10^3/\mu L$)	2.757 ± 1.272	1.969 ± 0.514	0.0004* [MWU]
C reactive proteins (mg/L)	104.56 ± 87.68	3.53 ± 1.8	<0.0001* [MWU]
Albumin (g/dL)	3.83 ± 0.49	4.19 ± 0.48	0.0024* [MWU]
Cardiac markers			
Troponin I (ng/mL)	6.54 ± 4.04	0.02 ± 0.01	<0.0001* [MWU]
Creatine Kinase MB (ng/mL)	55.19 ± 21.4	2.2 ± 1.54	<0.0001* [MWU]
Lipid profile			
Cholesterol (mg/dl)	152.71 ± 41.82	177.07 ± 17.13	0.0001* [MWU]
Triglycerides (mg/dl)	109.38 ± 52.45	125.93 ± 15.48	0.0002* [MWU]
High-density lipoprotein (mg/dl)	56.52 ± 12.62	51.2 ± 6.17	0.0002* [MWU]
Low density lipoprotein (mg/dl)	128.92 ± 30.07	98.6 ± 19.43	<0.0001* [MWU]
Very low-density lipoprotein (mg/dl)	28.92 ± 11.01	21.3 ± 11.59	0.0018* [MWU]
Hematologic inflammatory indices and ratios			
Mean platelet volume/platelet count ratio	0.04 ± 0.03	0.04 ± 0.01	0.99 [MWU]
Platelet mass index (fl/ml)	2.82 ± 0.44	1.94 ± 0.33	0.0001* [MWU]
White blood cells/mean platelet volume	1.2 ± 0.45	0.88 ± 0.22	0.0001* [MWU]
Red blood cells distribution width/Platelet	35.79 ± 13.65	55.67 ± 7.47	0.0001* [MWU]
Neutrophil to lymphocyte Ratio	2.34 ± 0.8	2.41 ± 0.49	0.1931 [MWU]
Platelet to lymphocyte ratio (PLR)	130.62 ± 68.88	126.09 ± 35.46	0.0092* [MWU]
Lymphocyte to C-reactive protein ratio	0.12 ± 0.17	0.83 ± 0.62	<0.0001* [MWU]
Albumin/C-reactive protein ratio	0.13 ± 0.16	1.8 ± 1.34	<0.0001* [MWU]
Platelet to C-reactive protein ratio	8.59 ± 9.47	99.62 ± 72.41	<0.0001* [MWU]
Systemic immune-inflammation index ($\times 10^9/L$)	720.28 ± 408.49	557.83 ± 120.4	0.0338* [MWU]

Platelet^2/ monocyte*neutrophil ratio (P2/MS $\times 10^{10}$)	141.98 ± 50.72	94.03 ± 43	<0.0001* [MWU]
Neutrophil platelets score (NPS)			
0: Neutrophils ≤ 7.5 & platelets ≤ 400	79 (79%)	27 (90%)	0.5322 [χ2]
1: Neutrophils > 7.5 or platelets > 400	21 (21%)	3 (10%)	
2: Neutrophils >7.5 & platelets >400	5 (5%)	0 (0%)	
Prognostic index (PI)			
0: CRP (≤10) & WBC (≤ 11,000)	11 (11%)	26 (86.67%)	< 0.0001* [χ2]
1: CRP (≤10) & WBC (> 11,000)	25 (25%)	4 (13.33%)	
1: CRP (>10) & WBC (≤ 11,000)	18 (18%)	0 (0%)	
2: CRP (>10) & WBC (> 11,000)	46 (46%)	0 (0%)	

*: significant; MWU: Mann-Whitney U test; f: χ2: Chi-square test.

ACS cases with EF < 50% showed no significant differences than those with EF > 50% in age, sex, family history, special habits, associated comorbidity, SBP, DBP, heart rate, anthropometrics measurements, and ECG findings (all P > 0.05). (**Table 3).**

Table 3. Basal demographic and clinical data in ACS patients regarding LVEF

Variables	ACS patients with LVEF		P-value
	< 50% (N = 53)	**> 50% (N = 47)**	
Age (Years) Mean ± SD	49.21 ± 10.2	51.77 ± 12.95	0.2846 [MWU]
Sex, N (%)			
• Male	28 (52.83%)	28 (59.57%)	0.5026 [χ2]
• Female	25 (47.17%)	19 (40.43%)	0.5026 [χ2]
Family history of CAD, N (%)			
• Positive	5 (9.43%)	6 (12.77%)	0.5995 [χ2]
• Negative	48 (90.57%)	41 (87.23%)	0.5995 [χ2]
Smoking, N (%)	6 (11.32%)	5 (10.64%)	0.9144 [χ2]
• Non-smoker	34 (64.15%)	34 (72.34%)	0.386 [χ2]
• Smoker	16 (30.19%)	10 (21.28%)	0.3154 [χ2]
• Ex-smoker	3 (5.66%)	3 (6.38%)	0.8808 [χ2]
Alcohol drinking, N (%)			
• Alcoholic	2 (3.77%)	0 (0%)	0.4968 [f]
• Non-alcoholic	51 (96.23%)	47 (100%)	0.1821 [χ2]
Comorbidities, N (%)			
• Diabetes Mellitus	12 (22.64%)	17 (36.17%)	0.1395 [χ2]
• Hypertension	21 (39.62%)	15 (31.91%)	0.428 [χ2]
• Hyperlipidemia	17 (32.08%)	13 (27.66%)	0.6347 [χ2]
• Atrial fibrillation	7 (13.21%)	6 (12.77%)	0.9484 [χ2]
• History of stroke	5 (9.43%)	4 (8.51%)	0.8737 [χ2]
Blood pressure			
• SBP (mmHg)	129.09 ± 12.28	125.4 ± 10.96	0.1419 [MWU]
• DBP (mmHg)	79.25 ± 8.47	77.32 ± 7.9	0.2557 [MWU]
Heart rate	84.74 ± 4.6	80.64 ± 9.67	0.0516 [MWU]
Anthropometrics			
• Weight (Kg)	71.11 ± 7.91	72.91 ± 9.77	0.3295 [MWU]

• Height (cm)	164.92 ± 6.04	165.55 ± 5.84	0.5867 [MWU]
BMI (Kg/m²)	26.31 ± 3.91	26.71 ± 4.09	0.7246 [MWU]
ECG			
• Normal ECG	1 (1.89%)	4 (8.51%)	0.1319 [χ2]
• ST elevation	13 (24.53%)	18 (38.3%)	0.1401 [χ2]
• ST depression	36 (67.92%)	23 (48.94%)	0.0548 [χ2]
• Inverted T Wave	3 (5.66%)	2 (4.26%)	0.7506 [χ2]

*: significant; f: Fisher exact test; MWU: Mann-Whitney U test; χ2: Chi-square test; CAD: coronary artery disease; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; ECG: Electrocardiogram; BMI: Body Mass Index.

ACS patients with EF < 50% had significantly higher mean platelet count and platelet distribution width (P < 0.0001), CRP levels (P < 0.0001), cardiac markers troponin (P = 0.001) and CK-MB (P = 0.0151), but lower MPV and lymphocyte count (P < 0.0001 and P = 0.0297). There were no significant differences in Hb, serum albumin, or lipid profile levels (P > 0.05).

ACS patients with LVEF < 50% showed increased inflammatory indices, including WBCs/MPV ratio, PLR (P < 0.0001), NLR (P = 0.0309), SII (P < 0.0001), and P2/MS (P = 0.004), and had NPS (P = 0.0278) and PI (P < 0.0001). However, significantly lower RDW/platelet, MPV/platelet, albumin/CRP, and platelet/CRP ratios were observed (all P < 0.0001).(**Table.4**).

Table 4. Laboratory parameters for ACS patients regarding LVEF

Variables	ACS patients with LVEF		P-value
	< 50% (N = 53)	**> 50% (N = 47)**	
Hb (g/dl)	14 ± 1.42	13.81 ± 1.84	0.8846 [MWU]
Platelet count (×10³/µL)	336.962 ± 73.198	246.83 ± 54.308	<0.0001* [s.t.]
Mean platelet volume (MPV fL)	8.91 ± 2.72	11.92 ± 3.57	<0.0001* [s.t.]
Plateletcrit PCT (%)	0.29 ± 0.04	0.28 ± 0.05	0.5679 [s.t.]
Platelet distribution width (PDW %)	4.4 ± 0.96	3.51 ± 1.12	<0.0001* [s.t.]
WBCs (×10³/µL)	11.225 ± 3.485	11.718 ± 3.412	0.3249 [MWU]
Monocyte count (×10³/µL)	0.511 ± 0.221	0.5 ± 0.213	0.9945 [MWU]
Neutrophil count (×10³/µL)	5.825 ± 1.861	5.881 ± 1.963	0.7955 [MWU]
Eosinophil count (×10³/µL)	0.232 ± 0.169	0.232 ± 0.183	0.9119 [MWU]
Lymphocyte count (×10³/µL)	2.551 ± 1.211	2.989 ± 1.298	0.0297* [MWU]
CRP (mg/L)	148.98 ± 89.11	54.47 ± 51.67	<0.0001* [s.t]
Albumin (g/dL)	3.9 ± 0.46	3.76 ± 0.52	0.1737 [s.t]
Cardiac markers			
Troponin I (ng/mL)	7.85 ± 3.9	5.06 ± 3.67	0.001* [s.t]
CK-MB (ng/mL)	58.75 ± 16.82	51.17 ± 24.99	0.0151* [s.t]
Lipid profile			
Cholesterol (mg/dl)	151.7 ± 44.96	153.85 ± 37.93	0.798 [MWU]
Triglycerides (mg/dl)	103.45 ± 58.91	116.06 ± 43.07	0.2298 [MWU]
High-density lipoprotein (mg/dl)	57.92 ± 12.21	54.94 ± 12.87	0.2379 [MWU]
Low-density lipoprotein (mg/dl)	126.42 ± 27.59	131.74 ± 32.41	0.3775 [MWU]
Very low-density lipoprotein (mg/dl)	28.57 ± 11.9	29.33 ± 9.88	0.7309 [MWU]

Hematologic inflammatory ratios			
Mean platelet volume/platelet count ratio	0.03 ± 0.04	0.05 ± 0.03	<0.0001* [s.t]
Platelet mass index (fl/ml)	2.85 ± 0.42	2.79 ± 0.47	0.5971 [s.t]
White blood cells/mean platelet volume	1.32 ± 0.45	1.05 ± 0.39	0.0009* [s.t]
Red blood cell distribution width/ platelet	31 ± 13.34	41.2 ± 11.87	<0.0001* [s.t]
Neutrophil to lymphocyte ratio (NLR)	2.51 ± 0.86	2.14 ± 0.67	0.0309* [s.t]
Platelet to lymphocyte ratio (PLR)	155.43 ± 65.45	102.65 ± 61.54	<0.0001* [s.t]
Lymphocyte to C-reactive protein ratio	0.05 ± 0.08	0.2 ± 0.21	<0.0001* [s.t]
Albumin/C-reactive protein ratio	0.06 ± 0.1	0.21 ± 0.17	<0.0001* [s.t]
Platelet to C-reactive protein ratio	4.49 ± 6.32	13.22 ± 10.27	<0.0001* [s.t]
Systemic immune-inflammation index ($\times 10^9$/L)	868.92 ± 440.12	552.67 ± 289.12	<0.0001* [s.t]
Platelet^2/ monocyte*neutrophil ratio (P^2/MS $\times 10^{10}$)	154.48 ± 48.52	127.88 ± 49.43	0.0036* [s.t]
Neutrophil platelets score (NPS)			
0: Neutrophils ≤ 7.5 & platelets ≤ 400	37 (69.81%)	42 (89.36%)	0.0278 [x]
1: Neutrophils > 7.5 or platelets > 400	16 (30.19%)	5 (10.64%)	
2: Neutrophils >7.5 & platelets > 400	4 (7.55%)	1 (2.13%)	
Prognostic index (PI)			
0: CRP (≤ 10) & WBC (≤ 11,000)	2 (3.77%)	9 (19.15%)	<0.0001* [s.t]
1: CRP (≤ 10) & WBC (> 11,000)	20 (37.74%)	5 (10.64%)	
1: CRP (> 10) & WBC (≤ 11,000)	3 (5.66%)	15 (31.91%)	
2: CRP (> 10) & WBC (> 11,000)	28 (52.83%)	18 (38.3%)	

*: significant; MWU: Mann-Whitney U test; s.t: Student's t-test; χ2: Chi-square test; Hb: Hemoglobin.

LVEF was significantly negatively correlated with platelet count, PDW, CRP, WBCs/MPV, SII (all P < 0.0001), PLR, and P2/MS (both P < 0.001), but positively correlated with MPV, MPV/platelet, RDW/platelet, lymphocyte/CRP, albumin/ CRP, and platelet/CRP ratios (all P < 0.0001), suggesting that the ACS disease process is associated with increased inflammation. (**Table.5).**

Table 5. Correlation between LVEF and different laboratory parameters

Variables	LVEF%	
Platelet count ($\times 10^3/\mu L$)	-0.607	< 0.0001*
Mean platelet volume (MPV fL)	0.465	< 0.0001*
Plateletcrit PCT (%)	-0.096	0.3418
Platelet distribution width (PDW %)	-0.417	< 0.0001*
C-reactive protein	-0.623	< 0.0001*
Mean platelet volume to platelet count (MPV/PLT)	0.375	0.0001*
Platelet mass index (PMI)	-0.096	0.342
White blood cells to mean platelet volume (WBCs/MPV)	-0.378	0.0001*
Red cell distribution width to platelet count (RDW/PLTs)	0.427	< 0.0001*
Neutrophil-to-lymphocyte ratio (NLR)	-0.176	0.0805
Platelet to lymphocyte ratio (PLR)	-0.329	0.001*
Lymphocyte to C-reactive protein ratio (L/CRP)	0.438	< 0.0001*
Albumin to C-reactive protein ratio (albumin/CRP)	0.501	< 0.0001*
Platelet to C-reactive protein ratio (P/CRP)	0.475	< 0.0001*

Systemic Immune-Inflammation Index (SII)	-0.399	< 0.0001*
Platelet2/ monocyte*neutrophil ratio (P^2/MS ×10^{10})	-0.313	0.001*

*: significant; r: Pearson correlation; LVEF: left ventricular ejection fraction.

Multivariable regression revealed that PDW (P = 0.019) and SII (P = 0.026) were independent positive predictors of LVEF, whereas CRP level (P = 0.038) was a significant independent negative predictor of LVEF. (**Table.6).**

Table 6. Multivariable regression analysis for predictors of ejection fraction

Variables	Unstandardized Coefficients B	Std. Error	OR	Test value	P-Value	95.0% Confidence Interval for B	
						Lower Bound	Upper Bound
(Constant)	55.24	25.84				3.83	106.65
PLTs	-0.12	0.1	-1.05	-1.25	0.215	-0.31	0.07
MPV	3.83	2.91	1.5	1.32	0.192	-1.96	9.63
PCT	641.44	829.26	3.18	0.77	0.441	-1008.53	2291.41
PDW	5.49	2.3	0.7	2.39	0.019*	0.91	10.06
CRP	-0.04	0.02	-0.37	-2.11	0.038*	-0.07	0
MPV/PLT	-237	178.66	-0.9	-1.33	0.188	-592.5	118.46
PMI	-69.28	83.66	-3.44	-0.83	0.41	-235.74	97.19
WBCs/MPV	-7.01	4.14	-0.35	-1.69	0.094	-15.24	1.23
RDW/PLTs	-0.04	0.22	-0.05	-0.17	0.864	-0.48	0.4
NLR	-7.74	4.93	-0.73	-1.57	0.12	-17.55	2.07
PLR	-0.04	0.03	-0.3	-1.33	0.188	-0.1	0.02
L/CRP	-10.71	11.85	-0.21	-0.9	0.369	-34.28	12.86
Albumin/CRP	-6.29	27.27	-0.11	-0.23	0.818	-60.55	47.97
P/CRP	0.07	0.39	0.08	0.19	0.853	-0.71	0.85
SII	0.03	0.01	1.47	2.27	0.026*	0	0.06
P2/MS	0.02	0.03	0.1	0.54	0.589	-0.05	0.08

*: significant; EF: ejection fraction; PLTs: platelets; MPV: mean platelet volume; PCT: plateletcrit; PDW: platelet distribution width; MPV/platelet count: Mean Platelet Volume to Platelet Count ratio; PMI: Platelet Mass Index; WBC/MPV: White Blood Cells to Mean Platelet Volume ratio; RDW/Platelet: Red Cell Distribution Width to Platelet ratio; EMR: Eosinophil to Monocyte Ratio; NLR: Neutrophil to Lymphocyte Ratio; MLR: Monocyte to Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; L/CRP ratio: Lymphocyte to C-reactive protein ratio; Albumin/CRP ratio: Albumin to C-reactive protein ratio; P/CRP ratio: Platelet to C-reactive protein ratio; SII: Systemic Immune-Inflammation Index; P2/MS: Platelet-to-monocyte ratio

Multivariable regression revealed that PDW (P = 0.046) was independent negative predictors of abnormal ECG findings. (**Table.7).**

Table 7. Multivariable regression analysis for predictors of abnormal ECG findings

Variables	Unstandardized Coefficients B	Std. Error	OR	Test value	P-Value	95.0% Confidence Interval for B	
						Lower Bound	Upper Bound
(Constant)	1.513	1.442	4.5403	1.05	0.297	-1.355	4.382
PLTs	0.005	0.005	1.005	1.003	0.319	-0.005	0.016
MPV	0.002	0.163	1.002	0.012	0.991	-0.321	0.325

PCT	45.796	46.267	7.437	0.99	0.325	-46.261	137.853
PDW	-0.26	0.128	0.7711	-2.025	0.046*	-0.515	-0.004
CRP	-0.001	0.001	0.999	-1.242	0.218	-0.003	0.001
MPV/PLT	1.242	9.968	3.4625	0.125	0.901	-18.592	21.075
PMI	-4.953	4.668	0.0071	-1.061	0.292	-14.24	4.335
WBCs/MPV	0.295	0.231	1.3431	1.276	0.206	-0.165	0.754
RDW/PLTs	0.007	0.012	1.007	0.537	0.593	-0.018	0.031
EMR	-0.443	0.314	0.6421	-1.411	0.162	-1.069	0.182
NLR	-0.289	0.275	0.749	-1.05	0.297	-0.836	0.258
MLR	1.291	1.268	3.6364	1.018	0.312	-1.233	3.815
PLR	0.001	0.002	1.001	0.496	0.621	-0.002	0.004
L/CRP	-0.423	0.661	0.6551	-0.64	0.524	-1.738	0.892
Albumin/CRP	0.18	1.521	1.1972	0.119	0.906	-2.847	3.208
P/CRP	-0.015	0.022	0.9851	-0.688	0.494	-0.059	0.029
SII	0	0.001	1	0.176	0.86	-0.001	0.002
P2/MS	-0.001	0.002	0.999	-0.575	0.567	-0.005	0.003

*: significant; EF: ejection fraction; PLTs: platelets; MPV: mean platelet volume; PCT: plateletcrit; PDW: platelet distribution width; MPV/platelet count: Mean Platelet Volume to Platelet Count ratio; PMI: Platelet Mass Index; WBC/MPV: White Blood Cells to Mean Platelet Volume ratio; RDW/Platelet: Red Cell Distribution Width to Platelet ratio; EMR: Eosinophil to Monocyte Ratio; NLR: Neutrophil to Lymphocyte Ratio; MLR: Monocyte to Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; L/CRP ratio: Lymphocyte to C-reactive protein ratio; Albumin/CRP ratio: Albumin to C-reactive protein ratio; P/CRP ratio: Platelet to C-reactive protein ratio; SII: Systemic Immune-Inflammation Index; P2/MS: Platelet-to-monocyte ratio

Multivariable regression revealed that PDW ($P < 0.0001$) and MPV/PLT ($P = 0.0041$) were independent positive predictors of ST elevation. Whereas PLTs ($P = 0.0008$) and MPV ($P = 0.0029$) were significant independent negative predictor of LVEF. (**Table.8).**

Table 8. Multivariable regression analysis for predictors of ST elevation

Variables	Unstandardized Coefficients B	Std. Error	OR	Test value	P-Value	95.0% Confidence Interval for B	
						Lower Bound	Upper Bound
(Constant)	-0.3169	1.355				-3.0129	2.3791
PLTs	-0.0172	0.005	0.9829	-3.4707	0.0008*	-0.0271	-0.0074
MPV	-0.4693	0.1528	0.6254	-3.072	0.0029*	-0.7733	-0.1653
PCT	-21.5131	43.4879	0	-0.4947	0.6222	-108.04	65.0141
PDW	0.688	0.1206	1.9897	5.7043	<0.0001*	0.448	0.928
CRP	0.0003	0.0009	1.0003	0.2935	0.7699	-0.0016	0.0022

MPV/PLT	27.6425	9.3694	192.46	2.9503	0.0041*	9.0003	46.2846
PMI	4.4826	4.3875	88.4644	1.0217	0.31	-4.2472	13.2123
WBCs/MPV	0.1257	0.217	1.1339	0.579	0.5642	-0.3061	0.5574
RDW/PLTs	0.0126	0.0116	1.0127	1.0792	0.2837	-0.0106	0.0357
EMR	-0.0157	0.2954	0.9844	-0.0532	0.9577	-0.6034	0.572
NLR	-0.3826	0.2585	0.6821	-1.4801	0.1427	-0.8969	0.1317
MLR	-0.1666	1.1923	0.8465	-0.1397	0.8892	-2.5388	2.2057
PLR	-0.0013	0.0015	0.9987	-0.8447	0.4008	-0.0044	0.0018
L/CRP	-0.4345	0.6212	0.6476	-0.6995	0.4863	-1.6706	0.8015
Albumin/CRP	1.322	1.4301	3.7509	0.9244	0.358	-1.5234	4.1674
P/CRP	-0.0059	0.0206	0.9941	-0.2854	0.776	-0.0468	0.0351
SII	0.0011	0.0007	1.0011	1.5289	0.1302	-0.0003	0.0026
P2/MS	-0.0025	0.0017	0.9975	-1.4133	0.1614	-0.0059	0.001

*: significant; EF: ejection fraction; PLTs: platelets; MPV: mean platelet volume; PCT: plateletcrit; PDW: platelet distribution width; MPV/platelet count: Mean Platelet Volume to Platelet Count ratio; PMI: Platelet Mass Index; WBC/MPV: White Blood Cells to Mean Platelet Volume ratio; RDW/Platelet: Red Cell Distribution Width to Platelet ratio; EMR: Eosinophil to Monocyte Ratio; NLR: Neutrophil to Lymphocyte Ratio; MLR: Monocyte to Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; L/CRP ratio: Lymphocyte to C-reactive protein ratio; Albumin/CRP ratio: Albumin to C-reactive protein ratio; P/CRP ratio: Platelet to C-reactive protein ratio; SII: Systemic Immune-Inflammation Index; P2/MS: Platelet-to-monocyte ratio

Multivariable regression revealed that PLTs ($P < 0.0001$) and MPV ($P < 0.0001$) were independent positive predictors of ST depression. Whereas PDW ($P < 0.0001$), MPV/PLT ($P = 0.002$) and PMI ($P = 0.021$) were significant independent negative predictor of ST depression. (**Table. 9).**

Table 9. Multivariable regression analysis between incidence of ST-depression and platelet and inflammatory indices

Variables	**Unstandardized Coefficients B**	**Std. Error**	**OR**	**Test value**	**P-Value**	**95.0% Confidence Interval for B**	
						Lower Bound	**Upper Bound**
(Constant)	2.314	1.389	10.1148	1.666	0.1	-0.45	5.078
PLTs	0.026	0.005	1.0263	5.17	<0.0001*	0.016	0.036
MPV	0.596	0.157	1.8148	3.806	<0.0001*	0.284	0.908

PCT	72.255	44.584	221.1	1.621	0.109	-16.454	160.964
PDW	-1.101	0.124	0.3325	-8.907	<0.0001*	-1.347	-0.855
CRP	-0.001	0.001	0.999	-0.881	0.381	-0.003	0.001
MPV/PLT	-30.35	9.606	0	-3.16	0.002*	-49.462	-11.238
PMI	-10.554	4.498	0	-2.346	0.021*	-19.504	-1.604
WBCs/MPV	0.278	0.222	1.3205	1.249	0.215	-0.165	0.72
RDW/PLTs	-0.017	0.012	0.9831	-1.422	0.159	-0.041	0.007
EMR	-0.574	0.303	0.5633	-1.894	0.062	-1.176	0.029
NLR	0.137	0.265	1.1468	0.519	0.605	-0.39	0.665
MLR	1.374	1.222	3.9511	1.124	0.264	-1.058	3.806
PLR	0.002	0.002	1.002	1.382	0.171	-0.001	0.005
L/CRP	-0.327	0.637	0.7211	-0.514	0.609	-1.594	0.94
Albumin/CRP	-1.155	1.466	0.3151	-0.788	0.433	-4.072	1.762
P/CRP	-0.004	0.021	0.996	-0.174	0.862	-0.046	0.038
SII	-0.001	0.001	0.999	-1.536	0.128	-0.003	0
P2/MS	0	0.002	1	0.103	0.918	-0.003	0.004

*: significant; EF: ejection fraction; PLTs: platelets; MPV: mean platelet volume; PCT: plateletcrit; PDW: platelet distribution width; MPV/platelet count: Mean Platelet Volume to Platelet Count ratio; PMI: Platelet Mass Index; WBC/MPV: White Blood Cells to Mean Platelet Volume ratio; RDW/Platelet: Red Cell Distribution Width to Platelet ratio; EMR: Eosinophil to Monocyte Ratio; NLR: Neutrophil to Lymphocyte Ratio; MLR: Monocyte to Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; L/CRP ratio: Lymphocyte to C-reactive protein ratio; Albumin/CRP ratio: Albumin to C-reactive protein ratio; P/CRP ratio: Platelet to C-reactive protein ratio; SII: Systemic Immune-Inflammation Index; P2/MS: Platelet-to-monocyte ratio

Multivariable regression revealed that PDW (P = 0.028) was independent positive predictors of inverted T-wave. **(Table.10).**

Table 10. Multivariable regression analysis between incidence of inverted T-wave and platelet and inflammatory indices

Variables	Unstandardized Coefficients B	Std. Error	OR	Test value	P-Value	95.0% Confidence Interval for B	
						Lower Bound	Upper Bound
(Constant)	-0.484	0.771	0.6163	-0.628	0.532	-2.017	1.049
PLTs	-0.004	0.003	0.996	-1.341	0.184	-0.009	0.002
MPV	-0.125	0.087	0.8825	-1.436	0.155	-0.298	0.048
PCT	-4.946	24.735	0.0071	-0.2	0.842	-54.16	44.268
PDW	0.154	0.069	1.1665	2.239	0.028*	0.017	0.29
CRP	-0.001	0.001	0.999	-1.253	0.214	-0.002	0
MPV/PLT	3.949	5.329	51.8835	0.741	0.461	-6.654	14.552
PMI	1.119	2.495	3.0618	0.448	0.655	-3.846	6.084
WBCs/MPV	-0.109	0.123	0.8967	-0.882	0.381	-0.354	0.137

RDW/PLTs	0.011	0.007	1.0111	1.669	0.099	-0.002	0.024
EMR	0.146	0.168	1.1572	0.868	0.388	-0.188	0.48
NLR	-0.044	0.147	0.957	-0.297	0.767	-0.336	0.249
MLR	0.083	0.678	1.0865	0.123	0.902	-1.266	1.433
PLR	-1.00	0.001	0.9999	-0.078	0.938	-0.002	0.002
L/CRP	0.339	0.353	1.4035	0.959	0.34	-0.364	1.042
Albumin/CRP	0.013	0.813	1.0131	0.016	0.987	-1.605	1.632
P/CRP	-0.006	0.012	0.994	-0.47	0.639	-0.029	0.018
SII	0	0	1	0.411	0.682	-0.001	0.001
P2/MS	0.001	0.001	1.001	1.225	0.224	-0.001	0.003

*: significant; EF: ejection fraction; PLTs: platelets; MPV: mean platelet volume; PCT: plateletcrit; PDW: platelet distribution width; MPV/platelet count: Mean Platelet Volume to Platelet Count ratio; PMI: Platelet Mass Index; WBC/MPV: White Blood Cells to Mean Platelet Volume ratio; RDW/Platelet: Red Cell Distribution Width to Platelet ratio; EMR: Eosinophil to Monocyte Ratio; NLR: Neutrophil to Lymphocyte Ratio; MLR: Monocyte to Lymphocyte Ratio; PLR: Platelet to Lymphocyte Ratio; L/CRP ratio: Lymphocyte to C-reactive protein ratio; Albumin/CRP ratio: Albumin to C-reactive protein ratio; P/CRP ratio: Platelet to C-reactive protein ratio; SII: Systemic Immune-Inflammation Index; P2/MS: Platelet-to-monocyte ratio

Discussion

Our study included 130 participants, 100 ACS patients with AMI, and 30 controls with stable CAD. No significant differences were found in age, smoking status, alcohol use, or comorbidities such as diabetes, hypertension, and hyperlipidemia. 11% of ACS cases had a positive family history of CAD and 13% had atrial fibrillation, and 9% had a history of stroke, which suggests that cerebrovascular and cardiovascular diseases are linked through systemic inflammation, endothelial dysfunction, and prothrombotic states **(Fan et al., 2022).**

Our findings aligned with **Wahrenberg et al. (2020),** who found that 4.7% of 28,188 people had ACS, and a family history of CAD was associated with ACS regardless of age, gender, cardiovascular risk factors, or ECG results. Patients younger than 65 with a family history of early onset CAD showed a stronger correlation. Moreover, **Rashid et al. (2020)** found that 57% of 160 patients had ACS, with risk factors including hypertension (63%), smoking (44%), diabetes (38%), and dyslipidemia (31%), all $p < 0.05$. However, obesity (22%) and a history of stroke (12%) were not statistically significant ($p > 0.05$).

In our study, patients with ACS had higher systolic blood pressure than controls ($p < 0.0001$), whereas diastolic pressure, heart rate, and BMI were equal. ACS patients were shorter ($p = 0.0004$) and lighter than controls ($P = 0.0001$). ECG showed 31% ST elevation, 59% ST depression ($p = 0.0003$), and 5% normal, and 53% of patients had LVEF below 50% with a mean LVEF of 48.09 ± 8.93%. Our findings were consistent with **Bangalore et al. (2010)** and **Harjola et al. (2020)**.

In this study, patients with ACS had higher hemoglobin, RBC, HCT, MCHC, platelet count, MPV, PDW, PCT, WBCs count, and neutrophil count, indicating compensatory hematopoietic responses, with considerably decreased MCV, MCH, and RDW, indicating iron metabolism disruption (all $p < 0.0001$).

Increased platelet activity, WBC, and neutrophil counts in ACS demonstrate the complex interplay of inflammation, coagulation, and erythropoiesis **(Babes et al., 2021).** According to **Budzianowski et al. (2017),** both anemic and nonanemic patients have higher cardiovascular

morbidity and mortality rates when their RDW is 12% or higher.

Our study found that ACS patients had higher CRP, troponin I, CK-MB, LDH, and VLDL levels than controls, indicating inflammation, myocardial damage, and ischemic injury, but had lower albumin, total cholesterol, and triglycerides (all $p < 0.01$), and lower serum albumin indicating disease severity. Higher VLDL levels indicate atherogenesis. This was consistent with **He et al. (2010),** who reported that higher CRP levels significantly increased the risk of ACS, with moderate levels (3.1–10 mg/L) increasing risk by 40% and high levels (>10 mg/L) increasing risk by 118%. These findings highlight the complicated metabolic and inflammatory mechanisms that cause ACS **(Li et al., 2023).**

In this study, patients with ACS had higher PI, with scores of 1 and 2, and higher inflammatory indices, such as PMI, WBC-to-MPV, SII, P2/MS, and PLR, but lower RDW-to-platelet, lymphocyte-to-CRP, albumin-to-CRP, and platelet-to-CRP ratios than controls (all $p < 0.05$). This suggests increased inflammation and thrombotic activity and determines the disease severity **(Budzianowski et al., 2017).** These results aligned with **Li et al. (2017)** and **Emre et al. (2020).**

In this study, diabetes was more common in patients with ACS with LVEF < 50% (36.17% vs. 22.64%); nevertheless, anthropometric measurements, comorbidities, vital signs, ECG results, and demographics were similar to those of patients with LVEF > 50%. Heart failure results from diabetes-related cardiac alterations, such as endothelial inflammation and stiffness, mostly affecting diastolic function while preserving systolic function **(Shin et al., 2020).**

In this study, ACS patients with LVEF < 50% had higher platelet counts and PDW but lower MPV and lymphocyte counts than those with LVEF > 50%. This indicating increased platelet turnover, activation, and systemic inflammation. These findings are consistent with **Bekler et al. (2015) and Paolucci et al. (2024).** In contrast, **Adam et al. (2018)** reported higher MPV but lower platelet numbers in ACS patients with LVEF < 50%, signifying different platelet turnover and activation mechanisms.

In this study, CRP, troponin I, and CK-MB levels were significantly higher in ACS patients with LVEF < 50%, suggesting a connection between myocardial injury, elevated systemic inflammation, and heart failure development. This finding is consistent with **Athilingam et al. (2013) and Wettersten and Maisel (2015).**

In this study, ACS patients with EF < 50% had higher inflammatory indices (WBCs/MPV, RDW/platelet, NLR, and PLR), indicating enhanced systemic inflammation and immune activation. Moreover, LVEF was positively correlated with MPV, MPV/platelet, RDW/platelet, lymphocyte/CRP, albumin/CRP, and platelet/CRP ratios, while it was negatively correlated with platelet count, PDW, CRP, WBCs/MPV, SII, PLR, and P2/MS (all $P < 0.05$), suggesting that the ACS disease process is linked to enhanced systemic inflammation. These findings were in line with **Mongirdienė et al. (2021),** who found a negative relationship between MPV ($r = -0.311$, $p = 0.0001$), neutrophil and monocyte counts, and inflammatory markers such as CRP, fibrinogen, and LVEF. However, **Bolat et al. (2016)** revealed no correlation between LVEF and MPV/platelet ratio, suggesting platelet dynamics differ by patient type.

Regression analysis in this study revealed that PDW and SII were independent positive predictors of LVEF, whereas CRP (all $P = 0.05$) was a significant independent negative predictor. This aligns

with **Koller et al. (2014),** who found that among heart failure patients with preserved EF, CRP was a poorer predictor of death.

According to **Balci et al. (2023),** higher SII scores were associated with reduced cardiac output and LVEF, indicating that systemic inflammation plays a role in left ventricular dysfunction. **Neves et al. (2025)** revealed that PDW was a reliable predictor of LVEF and increased mortality risk in heart failure patients**.**

Similar to our study, **Jin et al. (2025)** found significant linear relationship between SII and coronary lesion severity ($P < 0.05$).

In our study, the PDW ($P < 0.0001$) and MPV/PLT ($P = 0.0041$) were positive indicators of ST elevation, while platelet count ($P = 0.0008$) and MPV ($P = 0.0029$) were significant negative indicators of LVEF. Furthermore, platelet count and MPV ($P < 0.0001$) were independent positive predictors of ST depression. Whereas PDW ($P < 0.0001$), MPV/platelet ($P = 0.002$), and PMI ($P = 0.021$) were significant independent negative predictors of ST depression.

In our study, higher platelet counts and MPV predicted ST-segment depression, indicating a prothrombotic state. Lower PDW, MPV/PLT ratio, and PMI were linked to reduced platelet reactivity and lower ischemic risk **(Machado et al., 2018).** This aligns with **Cetin et al. (2017)**, who found a significant positive correlation between PDW and STEMI, particularly in younger patients. PDW levels were higher in young STEMI patients. Conversely, **Alvitigala et al. (2018)** reported that STEMI patients had significantly higher MPV (8.22 fL vs. 7.74 fL, $p = 0.005$) and PDW (15.81 fL vs. 15.62 fL, $p = 0.007$) compared to controls. MPV and PDW showed a positive correlation ($r = 0.556$, $p = 0.0001$), while platelet count had a negative correlation.

In this study, multivariable regression revealed that PDW ($P = 0.028$) was an independent positive predictor of inverted T-wave. This was in harmony with **Kumar (2023),** who found that PDW is an independent prognostic marker in ACS and MI patients, suggesting a positive correlation with T-wave inversion. Higher PDW levels are associated with T-wave inversion in ACS patients.

Limitations: A cross-sectional, small sample size, and single-center design may limit generalizability. We used a single baseline measurement of hematologic and inflammatory indices and LV function across ACS subtypes, missing the potential dynamic changes. Confounding factors like medication use, comorbidities, and other biomarkers were not fully accounted for. Lastly, causality cannot be established, requiring prospective, multicenter studies for validation.

Conclusion

There was a significant association between hematologic inflammatory indices and LVEF in patients with ACS. Increased WBC/MPV, RDW/platelet, NLR, PLR, L/CRP, albumin/CRP, P/CRP, SII, and P2/MS ratios were associated with increased systemic inflammation and reduced LVEF. PDW and SII were independent positive predictors of LVEF, whereas CRP level was a significant negative predictor. Inflammatory and platelet markers are crucial for predicting cardiac function and disease severity, suggesting their potential application in risk stratification and prognosis evaluation.

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