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ORIGINAL RESEARCH

Haematological Indices in Acute Coronary Syndrome Patients in Ethiopia: A Comparative Cross-Sectional Study

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Background: Numerous biomarkers are used as diagnostic, prognostic, and predictive indicators of myocardial ischemia. The most commonly used biomarkers are cardiac troponin I (Tn-I) and creatinine kinase (CK-MB). However, in developing nations, their availability in primary care settings is extremely limited. In such situations, easily available assays such as complete blood count (CBC) should be investigated as prognostic indicators in individuals with acute coronary syndrome (ACS).

Objective: This study aimed to compare the pattern of haematological indices and blood cell ratios of ACS patients compared with apparently healthy controls.

Methods: Patients diagnosed with ACS were recruited consecutively between 01 May 2022 and 31 October 2023 at Jimma Medical Center (JMC). Biochemical analyses and complete blood counts were performed. Analysis of variance was performed to compare the continuous variables. Spearman correlation coefficient tests were performed to correlate hematologic parameters with high sensitive troponin-I (hs-Tn-I) levels.

Results: This study enrolled 220 participants (110 patients with ACS and age, sex, and place of residence matched 110 non-ACS controls). From ACS group 99 (90%) were diagnosed with ST-elevated myocardial infarction. The ACS group had a significantly greater mean platelet volume (MPV), white blood cell count, red cell distribution width (RDW), neutrophil-to-lymphocyte ratio, and platelet-to-lymphocyte ratio. The RDW ($r = 0.248$, $p = 0.009$) and MPV ($r = 0.245$, $p = 0.009$) were significantly positively correlated with hs-Tn-I levels in the ACS group. MPV, RDW, and monocyte count were significantly higher in non-survivor ACS patients ($p < 0.05$).

Conclusion: The significant differences observed in haematological parameters between individuals with ACS and healthy controls suggest the potential utility of these easily accessible and cost-effective diagnostics in predicting future morbidity and ACS risk. Incorporating these routine evaluations into clinical practice could enhance risk assessment and improve patient outcomes.

Keywords: acute coronary syndrome, haematological indices, ACS prognosis, mortality risk, Jimma medical center

Introduction

Acute coronary syndrome (ACS) is a collection of signs and symptoms induced by plaque rupture and platelet-rich coronary thrombosis. Thrombus produces partial or full coronary artery obstruction, resulting in myocardial ischemia and numerous clinical symptoms, ranging from unstable angina (UA) to myocardial infarction, which are common causes of mortality in developing nations.[1] Biomarkers can reflect the pathophysiological process of ACS, provide expressive information about prognosis, and support clinical decision-making without replicating any clinically available information. Numerous ACS biomarkers are currently available and clinically used as diagnostic, prognostic, or predictive indicators.[2] The most commonly used biomarkers are high-sensitivity troponin T or I (hs-cTn) and creatinine phosphokinase MB (CK-MB).[3] Numerous studies have shown a strong correlation between haematological parameters and the

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risk of unfavorable outcomes in patients with ACS because of systemic hypoxemia and inflammation linked to pathophysiological mechanisms. Depending on the severity of the injury, this can lead to circulatory failure and, ultimately, organic dysfunction.[4]

The inflammatory processes and increased myeloid cell activity linked to ACS resulted in a rise in erythropoietin levels. Haematological parameters in peripheral blood can be used to diagnose the condition. The onset and progression of atherosclerotic plaques are significantly influenced by low levels of chronic inflammation, which results in the instability of the plaque and the production of thrombus.[5]

Neutrophils are the first class of leukocytes to be found in damaged areas. Upon activation, a substantial quantity of inflammatory mediators are generated, which regulate the reaction to tissue damage by exhibiting proteolytic enzymes, hypoxic damage, and other mediators.[6] Cytokines (interleukin III, interleukin VI, and thrombopoietin) are essential for controlling megakaryocyte ploidy and platelet quantity during the formation of larger-volume platelets.[7] Leukocyte recruitment to atherosclerotic lesions is boosted by platelet–leukocyte interactions, which also excite neutrophils and lead to the formation of neutrophil extracellular traps, which bind platelets and accelerate atherogenesis and atherothrombosis.[8] A quick and easy way to determine a patient's inflammatory condition is to use the neutrophil-to-lymphocyte ratio (NLR). Its value has been demonstrated in the stratification of mortality in the majority of cardiac events, as well as in the prediction and marking of infectious or inflammatory diseases and problems following surgery.[9] The heterogeneity of red blood cell size is measured by red cell distribution width (RDW), which is derived from the RBC size distribution curves. It has been demonstrated that this measure can predict morbidity and death in a number of cardiovascular conditions, including acute myocardial infarction, stable coronary artery disease, and heart failure.[10,11] Additionally, RDW has been suggested as a stand-alone predictor of mortality for patients with non-ST-elevation myocardial infarction (NSTEMI).[12]

Despite the overwhelming number of studies on hematologic biomarkers and cardiovascular disease, there is a paucity of published comprehensive studies on the relevance and implications of haematological parameters for ACS in low-income settings in sub-Saharan Africa, particularly where anemia and infection due to parasites, bacteria, mycobacteria, and viruses are common and affect hematologic biomarkers. Thus, this study assessed the haematological indices between patients with ACS and an apparently healthy control group.

Materials and Methods

Research Design and Data Collection

This study was conducted in the cardiovascular unit of the Jimma Medical Center (JMC) for 18 consecutive months (May 1, 2022, to October 31, 2023). JMC is one of the largest and oldest teaching and referral hospital in southwest Ethiopia. An institution-based comparative cross-sectional study was implemented to determine the pattern of haematological indices among patients with ACS and an apparently healthy comparative group. Consecutive patients admitted with confirmed ACS (based on electrocardiography, cardiac biomarkers, and clinical symptoms) during the study period were included. Patients aged less than 18 years and those who had been diagnosed with a haematological disease, known malignancy, systemic inflammatory or autoimmune disease, thrombocytopenia before admission, secondary anemia, chronic liver disease, renal failure, immunosuppressive therapy, use of anticoagulant agents, and readmission after discharge were excluded. Age-, sex-, and place of residence-matched apparently healthy individuals (a person without sign and symptoms of ACS and free from any chronic diseases listed in the exclusion criteria) from the Jimma community, Jimma University workers, patient visitors, and patient attendants were recruited purposively in a 1–1 ratio as the non-ACS comparative group.

After obtaining written informed consent, the participant's medical records were assessed for the possibility of exclusion criteria. Then, face-to-face interviews were conducted with both groups using a structured questionnaire. Data on the demographic, health-related, clinical, and outcome status of patients with ACS were recorded. Venous blood samples were collected under aseptic conditions using a disposable syringe; 2 mL of it was placed in an ethylenediaminetetraacetic acid (EDTA) tube for complete blood count analysis and the remaining 2 mL in a vacuum tube (organ tube) for serum biochemical analysis. Trained laboratory professionals performed the complete blood count (CBC) and serum biochemical analyses. Complete blood counts, including hemoglobin, red blood cells (RBC), RDW, white blood cells (WBC), WBC differential

counts (neutrophils, lymphocytes, eosinophils, basophils, and monocytes), platelet count (PLC), mean platelet volume (MPV), Plateletcrit (PCT), and percentages, were analyzed using the Uni-CelDxH 800 Coulter Cellular Analysis System. The NLR, platelet-to-lymphocyte ratio (PLR), MPV-to-lymphocyte ratio (MPVLR), and WBC-to-MPV ratio (WMR) were calculated. A Roche Cobas Integra 400 analyzer was used to detect essential serum biochemical parameters, including creatinine levels and high-sensitivity troponin I. All measurements were performed within 30 minutes of blood collection. Blood sample quality and analyses were performed using the standard operating procedures.

Statistical Analysis

Stata-SE version 14 was used to analyze the data. The report format for continuous variables was mean ± SD. Categorical variables were described using percentages and definite values. A *t*-test or ANOVA was computed to compare continuous variables, and the Mann–Whitney U and Kruskal–Wallis tests were applied to assess values that were not normally distributed. Variables between categories were compared using the chi-square and Fisher's exact test. When the p-value was less than 0.05, differences were regarded as statistically significant. The correlation between the two continuous parameters was calculated using Spearman correlation coefficient.

Results

This study enrolled 220 participants (110 patients with ACS and 110 controls without ACS (non-ACS)). Of the 110 ACS patients, 74 (67.3%) were men, indicating a significantly higher population of men among ACS-admitted patients. In the study group, the final diagnoses for 99 (90%), 9 (8.2%), and 2 (1.8%) patients were STEMI, NSTEMI, and unstable angina (UA), respectively (Figure 1). The mean age of the ACS patients was 56.69 ± 11.9 years. The majority of ACS patients, 90 (81.8%), were discharged from the hospital with improvement, while 20 (18.2%) died (Figure 2). Some demographic and health-related factors were significantly different between the ACS patients and non-ACS controls. Age, sex, and place of residence were matched between the two groups. Higher levels of education and regular exercise participation were two characteristics that revealed notable disparities between the two groups (Table 1).

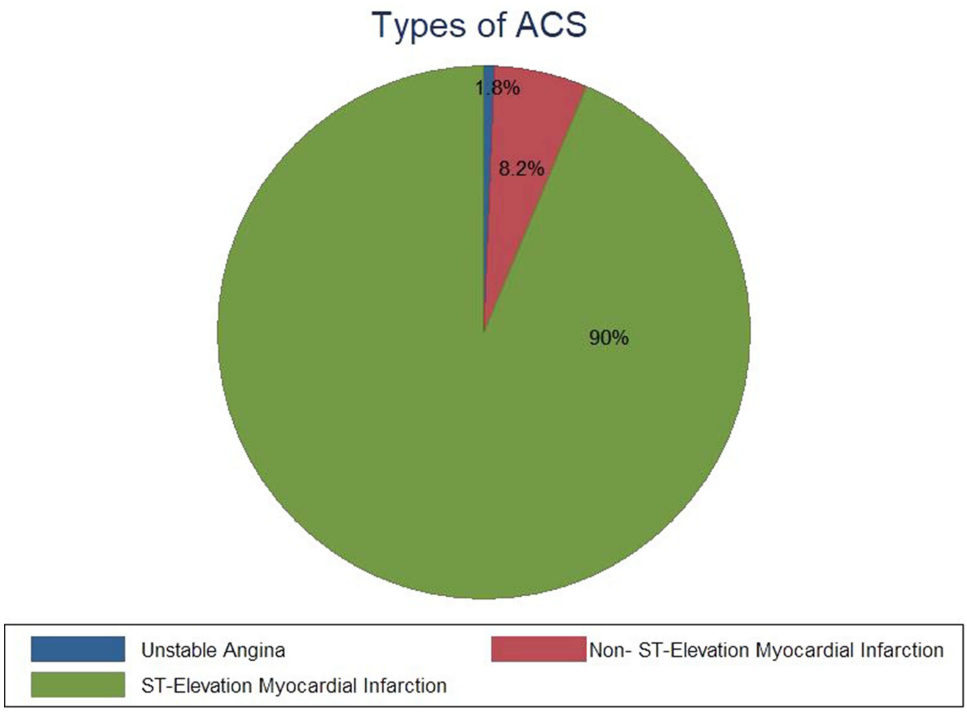

Figure 1 Types of acute coronary syndrome.

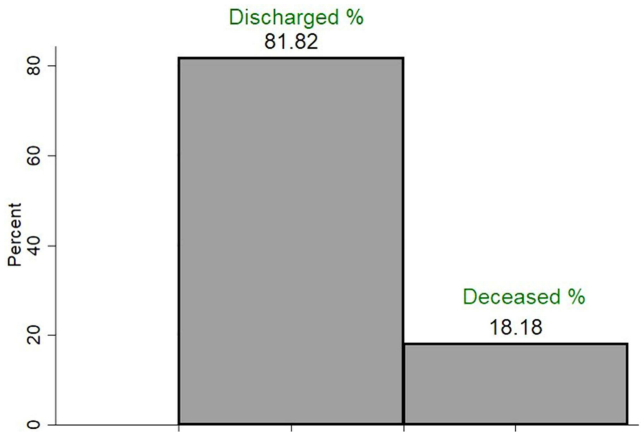

Figure 2 Acute coronary syndrome patients' outcome.

Analysis of differences in hematological indices and troponin levels between the ACS patients and non-ACS control groups showed significantly higher values in the parameters of WBC, MCHC, RDW, PLC, MPV, neutrophils, basophils, NLR, PLR, WBC-to-MPV ratio, MPVLR, hs-troponin I, and plateletcrit in the ACS group, whereas RBC, HGB, HCT, lymphocytes, and eosinophils were significantly higher in the non-ACS control groups (p <0.05). Table 2 displays the variations in each hematological parameter values between the two groups.

Correlations between haematological indices and highly sensitive troponin-I, the gold standard for ACS diagnosis, were computed in both study groups separately. There were no significant positive or negative correlations in the non-ACS control group, whereas RDW%, RDW-SD, and MPV were significantly positively correlated with hs-Tn-I (Table 3 and Figures 3 and 4).

Table 1 Demographics and Health-Related Characteristics of the Study Participants

Variables		ACS Case N (%)		Non-ACS Control N (%)		*p*-value
Age in Years	Mean (SD)	56.69 ± 11.91		56.5 ± 11.80		0.905
Sex	Male	74	67.27	74	67.27	1.000
	Female	36	32.73	36	32.73	
Residence	Urban	60	54.55	60	54.55	1.000
	Rural	50	45.45	50	45.45	
Educational status	Do not read and write	37	33.64	21	19.09	0.022
	Primary	25	22.73	38	34.55	
	Secondary	18	16.36	27	24.55	
	Tertiary	30	27.27	24	21.82	
Current cigarettes smoker	Yes	2	1.82	8	7.27	0.052
	No	108	98.18	102	92.73	
Regular exercise	Yes	25	22.73	94	85.45	0.000
	No	85	77.27	16	14.55	
Diabetes Mellitus	Yes	33	30.00	20	18.18	0.6855
	No	77	70.00	90	81.18	
Hypertension	Yes	50	45.45	25	22.73	0.2616
	No	60	54.54	85	77.27	
Family History of ACS	Yes	13	11.82	10	9.09	0.2554
	No	97	88.18	100	90.91	
BMI (kg/m^2)	*Mean (SD)*	24.0 ± 3.5		27.7 ± 29.1		0.194
WHR	*Mean (SD)*	1.0 ± 0.09		1.0 ± 0.1		0.100

Abbreviations: BMI, Body mass index; WHR, Waist-to-hip ratio.

Table 2 Comparison of Haematological Indices and Biochemical Markers Between ACS and Non-ACS Comparative Groups

Hematologic Indices	ACS Case Mean ±SD	Non-ACS Control Mean ±SD	*p*-value
White blood cell count 10^3 /μL, (Mean ±SD)	10.5±4.8	8.0±3.4	0.001
Red blood cell count 10^6/μL,(Mean ±SD)	4.7±0.9	5.0±0.6	0.007
Hemoglobin gm/dl, (Mean ±SD)	13.6±2.7	14.4±1.7	0.009
Hematocrit- % (Mean ±SD)	40.8±7.7	44.4±5.7	0.001
MCV fl, (Mean ±SD)	87.7±7.8	88.8±6.7	0.278
MCH pg, (Mean ±SD)	29.1±3.0	28.8±1.8	0.345
MCHC mg/dl, (Mean ±SD)	32.97±2.4	32.34±1.9	0.031
Red cell distribution width-CV (%)	19.5±2.2	14.4±1.8	0.001
RDW-SD fl, (Mean ±SD)	62.1±7.2	45.4±8.0	0.001
Platelet count 10^3 /μL, (Mean ±SD)	254.6±134.5	236.7±75.1	0.224
MPV fl, (Mean ±SD)	11.8±1.1	10.6±1.4	0.001
Neutrophil count 10^3 /μL (Mean ±SD)	8.1±4.4	5.2±3.2	0.001
Lymphocyte count 10^3 /μL (Mean ±SD)	1.2±0.7	2.0±0.8	0.001
Monocyte count 10^3 /μL (Mean ±SD)	0.6±0.5	0.5±0.3	0.060
Eosinophil count 10^3 /μL (Mean ±SD)	0.1±0.2	0.2±0.2	0.001
Basophil count 10^3 /μL (Mean ±SD)	0.07±0.1	0.01±0.06	0.001
Neutrophil to lymphocyte ratio (Mean ±SD)	12.0±22.9	3.4±5.2	0.001
Platelet to lymphocyte ratio (Mean ±SD)	313.0±457.2	135.4±92.0	0.001
WBC to MPV ratio (Mean ±SD)	0.9±0.4	0.8±0.4	0.030
MPV to lymphocyte ratio, (Mean ±SD)	15.2±18.3	6.2±4.2	0.001
Plateletcrit (%)	3.0±1.5	2.5±0.8	0.005
High Sensitive Troponin I (ng/L)	87.37±85.5	8.82±6.9	0.001

Abbreviations: MCV, Mean corpuscular volume; MCH, Mean Corpuscular Hemoglobin; MCHC, Mean corpuscular hemoglobin concentration; RDW, Red cell distribution width; MPV, Mean Platelet Volume.

Table 3 Correlations of Haematological Indices and Their Derivatives with High Sensitive Troponin-I

Hematologic Indices	ACS Case r	*p*-value	Non-ACS Control r	*p*-value
iWhite blood cell count 10^3 /μL, (Mean ±SD)	–0.024	0.804	–0.102	0.288
Red blood cell count 10^6/μL,(Mean ±SD)	0.136	0.157	–0.009	0.921
Hemoglobin gm/dl, (Mean ±SD)	0.064	0.508	0.033	0.725
Hematocrit- % (Mean ±SD)	0.068	0.483	–0.022	0.819
MCV fl, (Mean ±SD)	–0.125	0.191	0.011	0.903
MCH pg, (Mean ±SD)	–0.084	0.380	0.053	0.579
MCHC mg/dl, (Mean ±SD)	0.069	0.468	0.069	0.474
Red cell distribution width-CV (%)	0.248	0.009	–0.119	0.226
RDW-SD fl, (Mean ±SD)	0.189	0.047	–0.077	0.420
Platelet count 10^3 /μL, (Mean ±SD)	0.051	0.597	0.087	0.365
MPV fl, (Mean ±SD)	0.245	0.009	0.106	0.269
Neutrophil count 10^3 /μL (Mean ±SD)	0.003	0.969	–0.099	0.300
Lymphocyte count 10^3 /μL (Mean ±SD)	0.019	0.843	0.013	0.888
Monocyte count 10^3 /μL (Mean ±SD)	0.123	0.197	–0.073	0.446
Eosinophil count 10^3 /μL (Mean ±SD)	–0.068	0.478	–0.011	0.906
Basophil count 10^3 /μL (Mean ±SD)	–0.096	0.315	–0.098	0.310
Neutrophil to lymphocyte ratio (Mean ±SD)	–0.025	0.794	–0.134	0.162
Platelet to lymphocyte ratio (Mean ±SD)	–0.000	0.993	–0.070	0.465

(*Continued*)

Table 3 (Continued).

Hematologic Indices	ACS Case r	*p*-value	Non-ACS Control r	*p*-value
WBC to MPV ratio *(Mean ±SD)*	–0.078	0.413	–0.111	0.248
MPV to lymphocyte ratio, *(Mean ±SD)*	–0.018	0.844	–0.071	0.459
Plateletcrit (%)	0.113	0.237	0.110	0.254

Abbreviations: MCV, Mean corpuscular volume; MCH, Mean Corpuscular Hemoglobin; MCHC, Mean corpuscular hemoglobin concentration; RDW, Red cell distribution width; MPV, Mean Platelet Volume.

Haematological indices and biochemical markers based on patient outcomes were also compared. The mean differences in mean platelet volume (MPV), RDW%, RDW-SD, monocytes, creatinine, and hs-Tn-I were significantly higher in the non-survivor group, whereas the mean corpuscular volume (MCV) was significantly greater in the ACS survivor group (Table 4).

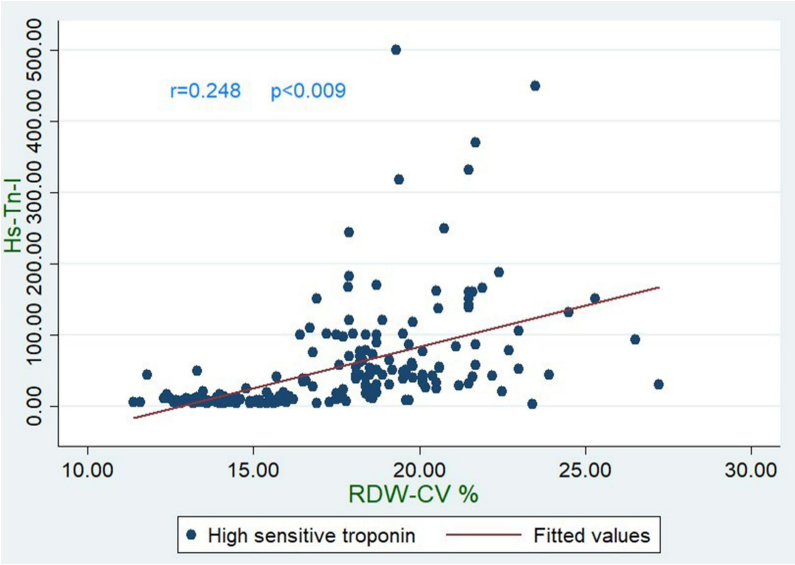

Figure 3 Correlation between RDW-CV % and high sensitive troponin-I.

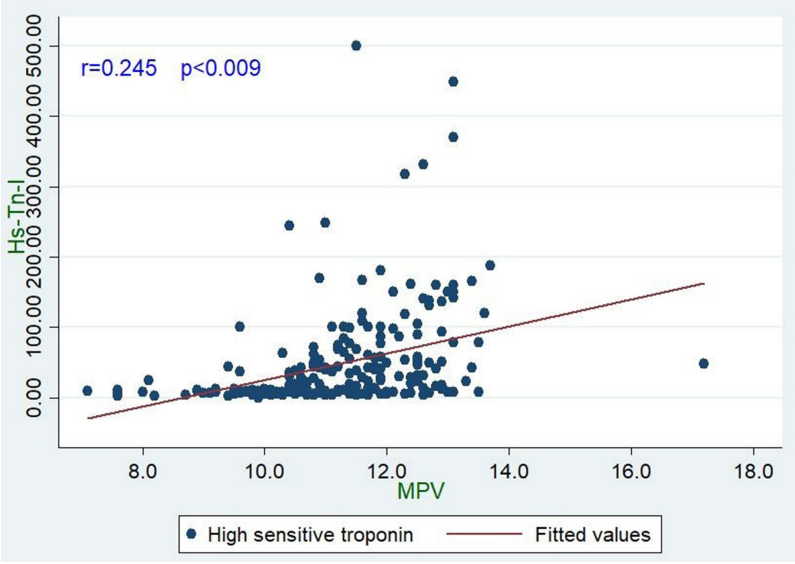

Figure 4 Correlation between MPV and high sensitive troponin-I.

Table 4 Comparison of Haematological Indices and Biochemical Markers Between ACS Survivors and Non-Survivor Groups

Hematologic Indices	ACS Survivors (N=90) Mean ± SD	ACS Non-Survivors (N=20) Mean ± SD	*p*-value
White blood cell count 10^3 /μL, (Mean ±SD)	10.198±4.8	12.035±4.5	0.122
Red blood cell count 10^6/μL,(Mean ±SD)	4.653±0.9	4.938±0.8	0.206
Hemoglobin gm/dl, (Mean ±SD)	13.551±2.6	13.716±2.7	0.805
Hematocrit- % (Mean ±SD)	40.780±7.8	41.159±7.4	0.843
MCV fl, (Mean ±SD)	88.671±7.9	83.515±6.3	0.007
MCH pg, (Mean ±SD)	29.356±3.1	27.968±2.0	0.058
MCHC mg/dl, (Mean ±SD)	32.906±2.4	33.280±1.9	0.526
Red cell distribution width-CV (%)	19.123±2.1	21.320±1.8	0.001
RDW-SD fl, (Mean ±SD)	60.976±7.0	67.164±5.8	0.001
Platelet count 10^3 /μL, (Mean ±SD)	256.598±143.2	245.750±87.3	0.745
MPV fl, (Mean ±SD)	11.658±1.1	12.425±0.9	0.005
Neutrophil count 10^3 /μL (Mean ±SD)	7.812±4.2	9.455±4.6	0.127
Lymphocyte count 10^3 /μL (Mean ±SD)	1.176±0.6	1.425±0.6	0.129
Monocyte count 10^3 /μL (Mean ±SD)	0.581±0.4	0.940±0.6	0.001
Eosinophil count 10^3 /μL (Mean ±SD)	0.120±0.1	0.195±0.2	0.075
Basophil count 10^3 /μL (Mean ±SD)	0.068±0.1	0.090±0.1	0.551
Neutrophil to lymphocyte ratio (Mean ±SD)	12.953±25.2	7.961±5.1	0.380
Platelet to lymphocyte ratio (Mean ±SD)	333.729±495.6	219.786±193.2	0.315
WBC to MPV ratio (Mean ±SD)	0.889±0.4	0.973±0.3	0.442
MPV to lymphocyte ratio, (Mean ±SD)	16.224±19.9	10.447±4.8	0.202
Plateletcrit (%)	2.970±1.6	3.061±1.1	0.811
Creatinine (mg/dL)	1.150±0.6	2.086±1.7	0.001
High Sensitive Troponin I (ng/L)	65.921±50.8	183.895±133.5	0.001

Abbreviations: MCV, Mean corpuscular volume; MCH, Mean Corpuscular Hemoglobin; MCHC, Mean corpuscular hemoglobin concentration; RDW, Red cell distribution width; MPV, Mean Platelet Volume.

Discussion

This study focused on the role of haematological indices and their derivatives in patients with ACS and an apparently healthy non-ACS comparative group, which was the first to be performed in the study area. Baseline characteristics for both groups were analyzed: ACS was dominated by 74 males (67.3%), and the mean age was 56.69 ± 11.91 years, which is less than 60 years. This outcome is in line with the findings of a prior Makassar research.[13] In fact, compared to other regions like Europe, African countries had younger age morbidity and death from non-communicable diseases, mainly cardiovascular disease.[14] On the other hand, according to Dai et al, patients older than 65 account for almost 60% of ACS hospital beds.[15] The quick changes in epidemiology that have occurred in African nations could be the cause of this discrepancy.

In this study, WBC, MCHC, RDW%, RDW-SD, PLC, MPV, neutrophils, basophils, NLR, PLR, WBC-to-MPV ratio, MPV-to-lymphocyte ratio, troponin-I, and plateletcrit were significantly higher in the ACS group, whereas RBC, HGB, HCT, lymphocytes, and eosinophils were significantly higher in the non-ACS control groups (p <0.05). This outcome is consistent with earlier research that found a significant increase in the MCHC value in ACS patients as compared to healthy controls.[16] In contrast, a study conducted in India showed no substantial differences in RDW and HCT between patients with ACS and healthy controls.[17] According to Koorts AM. et al, patients with ACS have a complicated interplay affecting their red blood cell profile that involves inflammation, iron metabolism, and anemia. The body lowers serum iron levels during inflammation by regulating macrophages and duodenal absorption.[18] Our findings support the hypothesis that people diagnosed with acute MI have higher MPV. Cameron et al demonstrated that not only does mean platelet volume correlate with more ischemic events, but MPV also remained specifically high for several weeks after infarction.[19] Furthermore, Alvitigala et al found that, in comparison to the healthy control group, STEMI patients had considerably higher mean MPV and PDW.[20]

Troponin is generally known as a cardiac biomarker elevated during ischemia. Cardiac troponin levels begin to elevate approximately 2 or 3 hours after myocardial injury. Correlations between haematological indices and highly sensitive troponin-I, the gold standard for ACS diagnosis, were computed in both study groups separately. Accordingly, RDW%, RDW-SD, and MPV were significantly positively correlated with hs-Tn-I (r = 0.248, 0.189, and 0.245, respectively; $p < 0.05$), whereas there were no significant positive or negative correlations in the non-ACS control group. According to research by Lippi et al, the sensitivity of cardiac troponin levels for detecting ACS increased from 94% to 99% when cardiac troponin and RDW were measured together at admission.[21] According to a different study, RDW strongly predicted acute myocardial infarction in female patients.[22] RDW is a low-cost, easy-to-use laboratory measurement technique that can predict ACS with a reasonable degree of diagnostic accuracy. Baseline RDW assessment appears effective for predicting myocardial damage at an earlier time point, given that the unique kinetics of troponin in the injured myocardium restricts its utility within 2–4 hours of symptom onset.

Previous research has demonstrated that patients with ACS have a greater MPV, which is consistent with our findings.[23–25] Due to platelet metabolic and enzymatic activity, an elevated MPV is associated with a number of cardiovascular risks as well as increased thrombogenicity.[26,27] In contrast to our findings, a study done by Kevin Luke et al showed that patients with ACS had significantly lower MPV than those with SCAD.[28] When predicting ACS, RDW and MPV should be taken into account in addition to traditional cardiac indicators. They should also be used as a reference when choosing the best course of treatment.

In this study, haematological indices and biochemical markers were compared based on the outcomes. The mean differences in MPV, RDW, monocyte count, creatinine, and hs-Tn-I were substantially greater among non-survivors, whereas the mean corpuscular volume (MCV) was higher among ACS survivors. This result is consistent with prior research, as Shahin et al, found that patients who passed away from MI had substantially greater MPV.[29] Małyszczak et al showed that both low and high MPV are significantly associated with a higher 5-year mortality rate than normal MPV cases.[30] Consistent with our findings, Raised RDW is a valuable indicator for morbidity and death in heart failure patients, according to Felker's research.[31] According to Tonelli et al, mortality risk was correlated with greater RDW levels in those who had previously experienced MI but did not exhibit symptoms of heart failure.[32]

Furthermore, research demonstrated a link between an increased RDW and a greater death risk in myocardial infarction patients.[10,33–35] Circulating monocytes interact largely with endothelial cells and platelets, aggravating prothrombotic and inflammatory pathways, and serving as a source of numerous cytokines and chemicals.[36] We measured creatinine and hs-troponin concentrations, and, as expected, mean creatinine and hs-troponin levels were significantly higher in the non-survivor group. Our Results are consistent with earlier research, which showed that creatinine clearance is a significant independent predictor of severe bleeding and in-hospital death in ACS patients.[37–39] Our study showed rising hs-Troponin I levels among non-survivors of ACS. Since levels of troponin and injured myocardial mass are strongly correlated, patients with ACS typically have greater troponin levels. Increased troponin levels are also an indicator of poor outcomes.[40] According to Kanani et al, greater hs-Troponin I readings were significantly linked to worse outcomes in the emergency room for both sexes; however, females had a higher inpatient mortality rate.[41]

Strengths and Limitations

To highlight the strength of this study, we extracted various types of information, such as cardiac parameters, hematologic indices, and biochemical markers. We examined how these variables were related to the clinical diagnoses and outcomes.

It is important to acknowledge the limitations of our investigation. Firstly, the study was cross-sectional, meaning that we may require additional time to observe and monitor changes and their impacts on the outcome. Additionally, the study involved a relatively small group of patients. To draw more conclusive results, we recommend a prospective cohort study with extended follow-up and a larger sample size. Such study should encompass a thorough analysis of cardiac, hematologic, and metabolic parameters, as well as consider various treatment modalities for patients with ACS.

Conclusion

This study explored the haematological profiles and their association with clinical outcomes in patients with ACS compared to healthy controls. Significant differences in haematological indices were observed between the groups, highlighting the potential of these markers in ACS diagnosis and prognosis. Correlations with highly sensitive troponin-I further emphasized the diagnostic

utility of parameters like RDW and MPV. Notably, differences in these markers also reflected outcomes, with non-survivors exhibiting distinct profiles. These findings underscore the importance of haematological indices in ACS management and prognosis, offering valuable insights for clinical practice and future research directions.

Abbreviations

ACS, Acute Coronary Syndrome; AMI, Acute Myocardial Infarction; GRACE, Global Registry of Acute Coronary Events; NSTEMI, Non-ST Elevation Myocardial Infarction; STEMI, ST Elevation Myocardial Infarction.

Data Sharing Statement

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available because it contain information that could compromise the privacy of research participants.

Ethical Approval and Informed Consents

The Jimma University Institutional Research Board (IRB) reviewed and approved the study protocol in accordance with the Declaration of Helsinki, assigning it the number IHRPGD/554/2022. Participants in the study were informed of the purpose of the investigation and the importance of their participation (or, in the event that the patient is unable of communicating or providing consent, their family member or caregiver). Written informed consent was obtained from each participant prior to any data collection. We certify that this article conforms to all applicable local, state, federal, and international laws pertaining to consent and privacy.

Acknowledgments

Jimma University and Jimma Medical Center.

Disclosure

The authors report no conflicts of interest in this work.

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