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Platelet Indices in Acute Myocardial Infarction- An Observational Study

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Abstract

Background: Platelet volume indices (PVI) such as mean platelet volume (MPV), platelet distribution width (PDW), and plateletcrit (PCT) have been implicated in the pathogenesis and prognosis of acute coronary syndromes (ACS). This study aimed to evaluate the diagnostic and prognostic utility of PVI in ACS among the Kashmiri population. ***Methods:*** A prospective observational study was conducted at GMC Srinagar from October 2022 to April 2024, enrolling 570 ACS patients and 285 age- and sex-matched healthy controls. PVI were compared between cases and controls, as well as among ACS subtypes: STEMI, NSTEMI, and unstable angina. Prognostic value was assessed for in-hospital and 30-day mortality. ***Results:*** MPV, PDW, and PCT were significantly higher in ACS patients compared to controls ($p<0.001$ for all). STEMI patients had significantly higher MPV and PDW compared to NSTEMI and unstable angina groups. PCT did not differ significantly among ACS subtypes. Multivariate analysis identified age ≥60 years, STEMI presentation, and elevated PDW as independent predictors of 30-day mortality. Platelet count did not differ significantly between cases and controls or among ACS subtypes. ***Conclusion:*** Elevated MPV, PDW, and PCT are associated with ACS and can be measured easily through routine hematological analysis. PDW, in particular, has prognostic significance for short-term mortality. PVI offer a simple, cost-effective adjunct for early detection, risk stratification, and prognostication in ACS.

Keywords: ***Acute coronary syndrome, mean platelet volume, platelet distribution width, plateletcrit, myocardial infarction, STEMI, NSTEMI, unstable angina, prognosis, Kashmir.***

Introduction

A cute coronary syndrome involves a spectrum of coronary artery disease from unstable angina to myocardial infarction. Acute myocardial infarction is an event of myocardial necrosis caused by an unstable ischemic syndrome [1]. Acute coronary syndrome requires a rise or fall in cardiac troponin (cTn) level (or another biomarker if cTn is not available), or both, accompanied by clinical evidence of ischaemia (i.e.,symptoms, ECG changes, supportive ECG or other imaging findings, or evidence of coronary thrombus) for diagnosis [2]. In practice, the disorder is diagnosed and assessed based on clinical evaluation, the electrocardiogram (ECG), biochemical testing, invasive and non-invasive imaging, and pathological evaluation. Globally, ischemic heart disease has become the leading contributor to the burden of disease as assessed based on disability-adjusted life-years [3]. The Global estimate of age-standardized CVD death rate of 272 per 1,00,000 population in India is higher than the global average of 235 per 1,00,000 population. Premature mortality in terms of years of life lost because of CVD in India has increased by 59% [4,5]. Acute myocardial infarction (AMI) defines cardiomyocyte necrosis in a clinical setting consistent with acute myocardial ischemia [6-8]. Acute myocardial infarction is further classified into six types: infarction due to coronary atherothrombosis (Type 1), infarction due to a supply-demand mismatch that is not the result of acute atherothrombosis (Type 2), infarction causing sudden death without the opportunity for biomarker or ECG confirmation (Type 3),infarction related to a percutaneous coronary intervention (PCI) (Type 4a), infarction related to thrombosis of a coronary stent (Type 4b), and infarction related to coronary- artery bypass grafting (CABG) (Type 5) [9]. Globally, ischemic heart disease has become the leading contributor to the burden of disease as assessed based on disability-adjusted life-years [10]. The usual initiating mechanism for acute myocardial infarction is rupture or erosion of a vulnerable, lipid-laden, atherosclerotic coronary plaque, resulting in exposure of circulating blood to highly thrombogenic core and matrix materials in the plaque [11]. A totally occluding thrombus typically leads to STEMI [12]. Partial occlusion, or occlusion in the presenceof collateral circulation, results in non-STEMI or unstable angina (i.e., an acute coronary syndrome without ST-segment elevation) [13]. In addition to aiding in diagnosis, different aspects of the electrocardiogram also provide prognostic information [14,15].Patients with NSTE-ACS and

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ST segment deviation-0.5 mV are at greater 1-year risk of death or MI than patients with T wave inversion or no ECG changes [16].Even when including cardiac biomarkers such as troponin, N-terminal pro-B type natriuretic peptide (NP), and C-reactive protein (CRP), the degree of ST-segment depression in patients with NSTE-ACS is the strongest prognostic variable for death or MI [17]. Overall, an elevated troponin is associated with roughly a 4-fold increase in the risk of death or recurrent MI compared with patients with a normal troponin concentration [18-20]. Even among patients with STEMI in whom biomarkers should not be used for diagnostic purposes, elevated troponin on admission is associated with worse outcomes [21,22], and peak troponin concentrations correlate with infarct size as determined by nuclear imaging [23]. Among patients with ACS, elevated levels of NP are strongly associated with clinical outcomes across the spectrum of ACS [24] including NSTE-ACS [25] and STEMI [26,27]. A variety of data collected from cardiac imaging can help to risk stratify patients after ACS. Assessing ischemia by a stress test in low- to intermediate-risk patients or in patients medically managed after MI is clearly indicated in practice Government Medical College, Srinagar 2 Introduction guidelines [28-30].

Platelets for long time have been implicated in the pathogenesis of cardiovascular diseases including atherosclerosis and its complications such as acute myocardial infarction, unstable angina, and sudden cardiac death. Platelet hyperactivity and local platelet activation have been found to play a role in acute coronary events [29]. Activated platelets are larger in size, and they can be measured by mean platelet volume (MPV) [31]. When platelets are larger, they become metabolically and enzymatically active. Platelet indices corresponds to functional status of platelets and is an emerging risk factor for atherothrombosis [32]. Platelet activation leads to a more spherical shape with increased platelet swelling and thereby leading to an increase in platelet mass and volume. Free arachidonic acid is also formed due to platelet activation, which may be converted into prostaglandins, also known as thromboxane A2. Platelet function and size correlate because larger platelets, produced from activated megakaryocytein thebone marrow, are likely to be more reactive than smaller platelets (normal) and secrete and express more mediators such as adhesive proteins, growth factors, and chemotactic and mitogenic factors (platelet factor 4, coagulation factors [factor V and factor XI], and cytokine like factors [interleukin1 and CD40 ligand] [33]. Larger platelets as measured by their volumes (MPV) may be useful markers in patients with ACS. Patients with increased MPV could be easily identified during routine haematological analysis. It could play an important role in early detection of acute coronary syndrome (ACS) and beneficial for preventive treatment and could be used as a screening test to differentiate the origin of chest pain along with other cardiac biomarkers. Unlike all other markers of platelet activation and reactivity, it is automatically calculated by most equipment for performing blood cell count [33]. Thus, to determine the platelet size through MPV is a simple, extremely inexpensive, and readily available measure in hospital and outpatient settings [33]. Analysis of PVI indicated MPV & PDW as an important risk factor for developing a myocardial infarction. This was along with the elevated cardiac enzymes levels [32]. MPV had higher sensitivity and specificity when compared to platelet count. MPV may be used as predictor for early detection of ACS and risk stratification when other cardiac biomarkers are negative. Patients with higher PCT and PDW are at higher risk of ACS. These patients can easily be identified during routine hematological examination and the patients could possibly benefit from preventive treatment [31]. This study has been conducted at several centres around the world and the value of different platelet volume indices as a diagnostic and prognostic marker of acute coronary syndrome has been already studied. The intention of this study was to prove the diagnostic and prognostic utility of these platelet volume indices in acute coronary syndrome in Kashmiri population and whether the same results will be found in this part of world as well. Our focus in this study was to prove the diagnostic and prognostic value of the platelet volume indices in acute coronary syndrome as these platelet volume indices are easily available at all centres around the world. Therefore the utility of platelet volume indices in acute coronary syndrome can be very helpful in the future.

Aim

To study therole of plateletvolumeindices(MPV, PDW, PCT) in acute coronary syndrome.

Objectives

Primary objective Primary objective was to compare the platelet indices in patients with acute coronary syndrome with age and sex matched healthy controls. Secondary objectives to compare these platelet volume indices between the different classes of acute coronary syndrome to check the prognostic value of platelet volume indices among different classes of acute coronary syndrome in terms of in hospital and 30 day mortality post hospitalization.

Materials And Methods

Our study was a Prospective observational study, conducted by the department of cardiology Super speciality hospital, GMC Srinagar from October 2022 to April 2024. We enrolled 570 patients over this period. Informed consent was taken from all the patients before enrolling them into the study. Further we took approval from institutional ethical committee before proceeding with this study. All the patients admitted to cardiology department Super speciality hospital with typical chest pain were subjected to detailed history and focused clinical examination. Relevant investigations like ECG and cardiac biomarker levels were analysed for confirmation of diagnosis. Myocardial necrosis was detected by rise and/or fall in cTn levels by conventional assays with at least one value above the 99th percentile URL.

Inclusion Criteria

Patients of acute coronary syndrome with ≥18 years of age with or without diabetes. Patients with Platelet count of more than 1 lakh per microlitre of blood.

Exclusion Criteria

Patients with underlying renal or liver disease. Patients with underlying coagulation disorders. Patients with underlying autoimmune or inflammatory disorders. Patients on oral anticoagulant drugs. Patients with COPD.

Statistical Methods

The recorded data was compiled and entered in a spreadsheet (Microsoft Excel) and then exported to data editor of SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were expressed as Mean±SD and categorical variables were summarized as frequencies and percentages. Graphically the data was presented by bar diagrams. Student's independent t-test and analysis of variance (ANOVA)was employed for comparing continuous variables. Chi-square test or Fisher's exact test, whichever appropriate, was applied for comparing categorical variables. Multivariate logistic regression analysis was applied to depict the independent variables of mortality. A P-value of less than 0.05was considered statistically significant.

Results

Among 570 ACS cases, mean age of cases was 58.2±11.39 years and mean age of controls was 57.8±11.98. Only 5.4% patients were 70 years of age. Among control group of 285 patients, 7.4% were 70 years of age. So, among both case and control, most common age group in our study belonged to 51-60 years of age. Among 570 ACS cases, 86.5% were males and 13.5% females. Among 285 controls 85.3% were males and 14.7% females. So, among both groups male sex was dominant. Among 570 ACS cases 71.4% were hypertensive, 68.9% were smokers, 42.6% were diabetic patients, 38.1% patients were having dyslipidaemia and 14.6% patients were having family history of acute coronary syndrome.

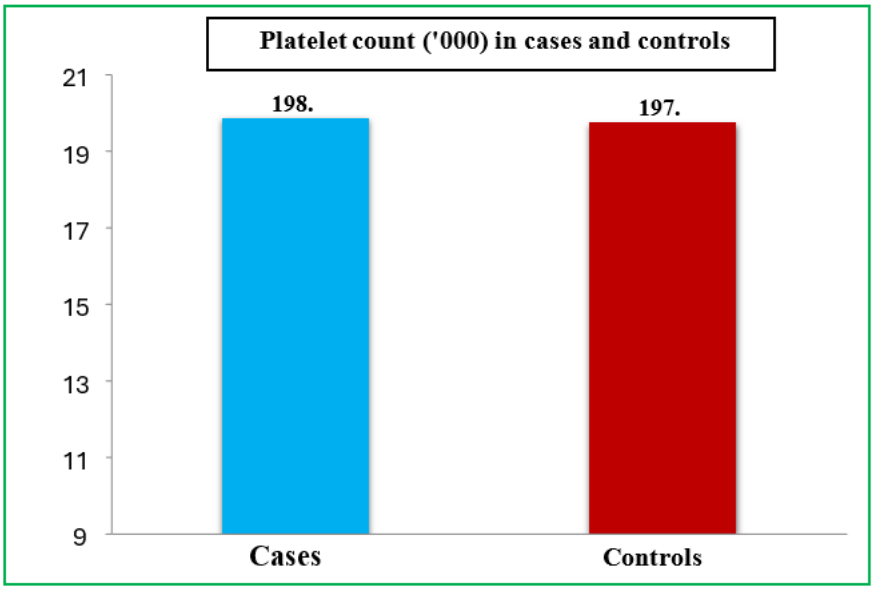

Figure 1: Shows the mean platelet count between cases and controls.

Figure 1 shows the comparison based on platelet count between cases and controls in our study. The mean platelet count of study cases was 198.4 42.35 and the mean platelet count of control group was 197.3 36.51 with a P value of 0.717.

Table 1:Comparison based on MPV(fl) in cases and controls

Group	N	Mean	SD	95% CI	P-value
Cases	570	11.2	1.45	11.08-11.32	
Controls	285	8.9	1.12	8.78-9.04	<0.001*

Table 1: Shows comparison based on mean MPV between cases and controls. The mean MPV of cases is 11.2□1.45fl and the mean MPV of controls is 8.9□1.12fl with a p value of <0.001.

Table 2:Comparison based on PDW(fL) in cases and controls

Group	N	Mean	SD	95%CI	P-value
Cases	570	18.2	1.52	18.02-18.29	
Controls	285	16.8	1.28	16.64-16.94	<0.001*

Table 2 shows comparison of PDW among cases and control group.The mean PDW ofcases is 18.2□1.52fL and the mean PDW of control group is 16.8□1.28fL with a p value of <0.001.

Table 3: Comparison based on Pct% in cases and controls

Group	N	Mean	SD	95% CI	P-value
Cases	570	0.231	0.042	0.227-0.234	
Controls	285	0.173	0.023	0.171-175	<0.001*

Table3 shows comparison based on mean Pct of cases and control group. The mean Pct of cases is 0.231□0.042% and the mean Pct of control group is 0.173□0.023% with a P value of <0.001.

Table 4: Comparison based on MPV(fL) between different classes of ACS

Group	N	Mean	SD	Comparison	P-value
GroupI	386	11.51	1.44	Ivs II	<0.001*
Group II	137	10.83	1.51	IIvsIII	<0.001*
Group III	47	9.21	1.36	Ivs III	<0.001*

Table 4 shows comparison based on mean MPV between different groups of ACS. The mean MPV of group ISTEMI patients is 11.51□1.44fL, mean MPV of group II NSTEMI patients is 10.83□1.51fL and the mean MPV of group III unstable angina patients is 9.21□1.36fL. Comparison made between mean MPV of group I and group II shows P value of <0.001, group II and group III shows P value of <0.001 and finally comparison between mean MPV of group I and group III shows P value of <0.001.

Table 5: Comparison based on PDW(fL) between different classes of ACS

Group	N	Mean	SD	Comparison	P-value
GroupI	386	18.37	1.48	Ivs II	0.002*
Group II	137	17.91	1.46	IIvsIII	0.001*
GroupIII	47	17.04	1.88	Ivs III	<0.001*

Table 5 shows comparison based on mean PDW values of different groups of ACS. The mean PDW of group 1 STEMI patients is 18.37□1.48fL, the mean PDW of group II NSTEMIpatients is17.91□1.46fL and the mean PDW of group III unstable angina patients is 17.04□1.88fL. Comparison made between group I and group II shows P value of 0.002, between group II and group III p value is 0.001 and between group I and group III shows P value of <0.001.

Table 6: Comparison based on Pct between different classes of ACS

Group	N	Mean	SD	Comparison	P-value
GroupI	386	0.229	0.0417	Ivs II	0.462
Group II	137	0.232	0.0421	IIvsIII	0.875
Group III	47	0.235	0.0424	IvsIII	0.519

Table 6 showing comparison based on Pct between different groups of ACS. The mean Pct of group 1 STEMI patients is 0.229□0.0417% and group II NSTEMI patients is 0.232□0.0421% and group III unstable angina patients is 0.235□0.0424. Comparison of group I with group II shows P value of 0.462, group II with group III shows P value of 0.875 and comparison of group I with group III shows P value of 0.519.

Table 7: Univariate analysis of 30-Day mortality

Parameter	Mortality [n=28]	Nomortality [n=542]	P-value
Age≥60	20 (71.4%)	254 (46.8%)	0.012*
Malegender	24 (85.7%)	469 (86.5%)	0.902
Smoker	25 (89.3%)	368 (67.9%)	0.017*
Hypertension	26 (92.9%)	382 (70.5%)	0.019*
DiabetesMellitus	15 (53.6%)	218 (40.2%)	0.162
Dyslipidaemia	12 (42.9%)	192 (35.4%)	0.423
FamilyhistoryofACS	6 (21.4%)	58 (10.7%)	0.079
STEMI	26 (92.9%)	360 (66.4%)	
NSTEMI	2 (7.1%)	128 (23.6%)	0.016*
UA	0 (0%)	47 (8.7%)	
PCI	11 (39.3%)	485 (89.5%)	
NonPCI	17 (60.7%)	57 (10.5%)	<0.001*
Troponin-I	13.1±7.18	11.4±9.63	0.361
Plateletcount ('000)	193.2±39.95	198.7±42.49	0.489
MPV	11.22±1.46	10.81±1.33	0.141
PDW	19.12±1.72	18.21±1.49	0.001*
Pct	0.225±0.041	0.231±0.042	0.447

Table 7 shows univariate analysis of different demographic and clinical variables of 30 day mortality among ACS cases. Total 28 of 570 patients had mortality that's 4.9%.

Table 8: Multivariate analysis depicting independent predictors of 30-DayMortality

Variable	OddsRatio	95% CI	P-value
Age≥ 60	2.53	2.03-3.48	0.008*
Smoker	0.84	0.64-1.29	0.372
Hypertension	1.04	0.74-1.57	0.431
STEMI	4.72	3.18-6.35	<0.001*
PCI	1.28	0.84-1.76	0.542
PDW	1.95	1.54-2.87	0.018*

Table8 shows multivariate logistic regression analysis of predictors of 30 day mortality suggesting age, STEMI and mean PDW were independent predictors of mortality.

Discussion

In our study, total number of patients were 570 and 285 age and sex matched controls were included in a 2:1 ratio from october 2022 to April 2024. Mean age of patients was 58.2 ± 11.39 and mean age of controls was 57.8 ± 11.98. Among cases 86.5 %were malesand13.5% were females. Among controls 85.3%were males and 14.7 % were females. Khandekar *et al*, included 210 cases in their study and the mean (SD) age of the patients was 53.9 (11.3) years with equal sex predilection [34].Birader SB *et al*, in a study carried out on 100 patients, the mean age of the participants was 55±10 years. Majority of the patient diagnosed as ACS belonged to 6th decade of life that is around 61.33% were males and 38.66% were females [35]. Our study was consistent with above studies and majority of our cases were in their sixth decade of life and majority belonged to male sex. Most common risk factor in our study population was hypertension seen in 71.4% (N=407), followed by smoking in 68.9% (N=393), diabetes mellitus 42.6% (N=243), dyslipidaemia 38.1% (N=217) and family history of ACS in 14.6 % (N=83). Khandekar *et al*. observed in their study that both

endogenous and exogenous risk factors such as smoking, diabetes mellitus, hypertension, hypercholesterolaemia, mental stress, and obesity–acting either singly or in combination-significantly increase the chances of developing coronary atherosclerosis. Most common risk factors in their study were hypertension and smoking[34]. Birader SB *et al* in their study among risk factors, smoking was highest in cases followed by diabetes mellitus and hypertension[35]. In our study the risk factor profile was consistent with above studies. The most common risk factor among study cases was hypertension followed by smoking and diabetes mellitus. Other less common risk factors were dyslipidaemia and family history of ACS. The mean platelet count of ACS cases was 198.4±42.35 103/µl controls mean platelet count was 197.3±36.51 103/µl, among non ACS, P value was 0.717. The mean platelet count of group 1 STEMI patients was 197.4±41.11 103/µl in group 2 NSTEMI patients it was 199.7±45.87 103/µl and in group 3 unstableangina patients, mean platelet count was 202.7±40.2 103/µl. When we compared mean platelet count of group 1 vs group 2 patients, p value was 0.586. When mean platelet count of group 2 vs group 3 patients was compared, p value was 0.691. And finally group 1 vs group 3 patients were compared based on mean platelet count, p value was 0.403. So mean platelet count among different groups of ACS patients was not statistically different. Platelet count is the basic important index. Some believe that platelet count and size are contrary to each other. It is assumed that the larger platelets will have a lower absolute count in order to maintain the total effectiveness of platelet mass [36]. We investigated the relationship between platelet count among ACS and non ACS patients. We found statistically no difference between two groups. Hendra *et al* by comparing platelet indices between the infarct group and non-infarct patients, reported that platelet count did not differ significantly between the two groups [37].

In another study, Boos *et al*. evaluated time-dependent changes of platelet indices in ACS patientsand found no significant changes in platelet count over time [38]. Khandekar *et al* evaluated platelet profile in ischemic spectrum among 210 Indian populations and found no significant differences in platelet count between different groups of ischemic patients [34]. Our study was consistent with above studies and found no statistically significant difference between ACS cases and control group and among different groups of ACS. In our study, the mean MPV of ACS patients was 11.2±1.45fL (11.08- 11.32 with 95% CI). The mean MPV of non ACS controls was 8.9±1.12fL (8.78-9.04 with 95% CI). The difference of MPV between ACS patients vs controls was statistically significant with p value of <0.001. Then the mean MPV of different ACS groups was compared among each other. The mean MPV of group 1 STEMI patients was 11.5±1.44fL. The mean MPV of group 2 NSTEMI patients was 10.83±1.51fL. The mean MPV of group 3 unstable angina patients was 9.21±1.36fL. The mean MPV of group 1 STEMI patients when compared to mean MPV of group 2 NSTEMI patients, difference was statistically significant with p value of <0.001.Then the mean MPV of group 2 NSTEMI patients was compared to mean MPV of group 3 unstable angina patients and the difference was statistically significant with p value of <0.001. Finally the mean MPV of group 1 STEMI patients was compared to mean MPV of group 3 Unstable angina patients and the difference was found to be statistically significant with P value of <0.001.

Mean platelet volume indirectly implies that platelet destruction and reproduction are relatively high. This can cause more activity than smaller-sized platelets. Patients with higher MPV are at higher risk of vascular occlusion.

Cameron *et al* demonstrated that not only mean platelet volume correlates with more ischemic events, but MPV also remained specifically high for several weeks after the infarction [39].

Martin *et al* compared platelet volume and distribution in 15 cases with myocardial infarction and 22 healthy people and theMean platelet volumewas found to be higher in the MI group even after six weeks after the infarction [40].

Birader SB *et al* in a study of 100 cases diagnosed with acute coronary syndrome and 50 controls concluded that MPV in ACS cases was higher compared to those with control group [41].

Jaya Manchanda *et al* in their prospective hospital based study including 175 cases of ACS and 175 controls concluded that MPV was raised in patients who have suffered an acute coronary event when compared with controls and those with unstable angina [42].

Our study was consistent with above mentioned studies and showed mean MPV was higher in ACS patients compared to non ACS control group. Among ACS patients mean MPV was higher in group 1 STEMI patients compared to Group 2 NSTEMI patients and group 3 unstable angina patients and group 2 NSTEMI patients had higher mean MPV compared to group 3 unstable angina patients.

In our study, the mean PDW of ACS cases was 18.2±1.52 fL (18.02-18.29with 95 % CI). The mean PDW of control group was 16.8±1.28 fL (16.64-16.94 with 95 % CI). The mean PDW of ACS group was higher compared to non ACS control group with a statistically significant difference with pvalue of <0.001.We also compared the PDW between different classes of acute coronary syndrome and we found that the mean PDW of group 1 STEMI patients was 18.37±1.48fL, mean PDW of group2 NSTEMI patients was 17.91±1.46 fL and mean PDW of group 3 unstable angina patients was 17.04±1.88fL.The mean PDW of group 1 STEMI was significantly higher than group 2 NSTEMI patients with a p value of 0.002.Compared to group 3 unstable angina patients, group 2 NSTEMI patients had significantly higher mean PDW with a p value of 0.001.Finally we compared mean PDW of group1 STEMI patients with mean PDW of group3 unstable angina patients and we found group 1 patients had significantly higher mean PDW compared to group 3 patients, with a p value of <0.001.

Platelet distribution width (PDW) reflects whether platelets are equal or different in size and shape. Platelets start to get larger when they are activated. Abdullah S. Assiri*et al* who examined 212 patients in a case-control study and found PDW was significantly higher in MI cases than incontrol groups [43]. Pervin S *et al*, Nandwani S *et al*, and Khandekar MM *et al*, described in their studies all platelet volume indices including MPV, PDW and PLCR were increased significantly in patients with ACS than controls [44,45,34]. During comparison of PDW and MPV, Pervin S *et al* found significant differences between the groups [45]. Our study showed similar results that is the mean PDW in ACS group was significantly higher than non ACS group. We also made comparison of mean PDW values between different groups of ACS and we found statistically significant difference. The mean PDW was significantly higher in group 1 compared to group 2, in group 2 it was significantly higher than group 3. Finally we compared mean PDW of group 1 with group 3 and it was significantly higher in group 1 patients. In our study, the mean platelet crit(Pct) of ACS patients was 0.231±0.042% (0.227-0.234 with 95 % CI) compared to non ACS control group whose mean Pct was 0.173±0.023% (0.171-0.175 with 95 % CI). Mean Pct in ACS patients was significantly higher compared to non ACS control groupwith p value of value of <0.001. We also compared Pct among different groups of ACS and we found that group 1 ACS patients had a mean Pct of 0.229±0.0417%, patients in group 2 NSTEMI had a mean Pct of 0.232±0.0421%,and patients in group 3 Unstable angina had a mean Pctof 0.235±0.0424%.Then we compared mean Pct of group 1 vs group 2 patients and the difference was statistically nonsignificant with a p

value of 0.462.We also compared mean Pct values of group 2 and group 3 patients and we found nonsignificant difference with a p value of 0.875.Finally we compared man Pct of group 1 STEMI patients with that of group 3 Unstable angina patients and this also showed non significant difference with p value of 0.519.

S P Akula *et al*, in a study 60 patients who presented with Acute Coronary Syndromes(ACS) there was a significant difference in Pct & PDW in patients with ACS compared to non ACS patients. Patients with ACS have higher values of Pct and PDW when compared to age matched controls (p=0.0001) [46].

Khode*et al*, in a comparative study of 128 subjects Increased MPV, PLC,P-LCR and PCT were observed in CAD group compared to controlgroup[47].

Conclusion

Our study intended to make a comparison of a simple and easy available investigation that's platelet volume indices (MPV, PDW, Pct) among acute coronary syndrome patients and age and sex matched healthy controls and then these platelet indices were compared among different classes of ACS. Our data suggests that the mean platelet volume, platelet distribution width and platelet crit was found to be increased in ACS group when compared to Healthy controls. Statistically significant difference in platelet volume indices was found to exist between cases (AMI group and Unstable angina group) and controls. The increased platelet volume indices contribute to the prethrombotic state in acute ischaemic syndromes such that larger platelets may play a specific role in infarction. Because larger platelets are haemostatically more active, the presence of larger platelets is probably a risk factor for developing coronary thrombosis and MI. Patients with larger platelets can easily beidentified during routine haematological analysis because PVI are generated as a part of routine automated blood counts. PVI is a feasible andeasyreliable test, that's helps in predicting prognosis among different acute coronary syndrome patients. Thus, in conclusion, PVI provides an important, simple, effortless, and cost-effective tool, which can be useful in predicting and prognosticating an acute coronary event.

Declarations

Ethical Approval

Not Applicable

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Conflict of interest

None declared

References

[1] Thygesen K, Alpert JS, Jaffe AS, *et al*. Third universal definition of myocardial infarction. JAm Coll Cardiol2012;60:1581-98.

[2] Thygesen K, Alpert JS, Jaffe AS, *et al*. Fourth universal definition of myocardial infarction (2018). Circulation 2018; 138: e618-51.

[3] Murray CJ, Barber RM, Foreman KJ, *et al*. Global, regional, andnational disability adjusted life years (DALYs) for 306 diseases andinjuries and healthy life expectancy (HALE) for 188 countries, 1990- 2013: quantifying the epidemiological transition. Lancet 2015; 386: 2145-91.

[4] Gupta R, Mohan I, Narula J. Trends in Coronary Heart Disease Epidemiology in India. Annals of Global Health. 2016;82:307-15.

[5] Prabhakaran D, Jeemon P, Roy A. Cardiovascular Diseases in India. Current Epidemiology and Future Directions. Circulation.2016;133:1605-20.

[6] Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, Bax JJ, Borger MA, Brotons C, Chew DP, Gencer B, Hasenfuss G, Kjeldsen K, Lancellotti P, Landmesser U, Mehilli J, Mukherjee D, Storey RF, Windecker S, ESC Scientific Document Group. 2015 ESC Guidelines for the management of acute coronary syndromes in patientspresenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). Eur Heart J 2016; 37:267_315.

[7] Ibanez B, James S, Agewall S, Antunes MJ, Bucciarelli-Ducci C,Bueno H, Caforio ALP, Crea F, Goudevenos JA, Halvorsen S, Hindricks G, Kastrati A, Lenzen MJ, Prescott E, Roffi M, Valgimigli M, Varenhorst C, Vranckx P, Widimsky P, ESC Scientific Document Group. 2017 ESC Guidelines for the management of acute myocardialinfarction in patients presenting with ST-segment elevation: The Task Force for the management of acute myocardial infarction in patients presenting with ST segment elevation of the European Society of Cardiology (ESC). Eur Heart J 2018;39:119_177.

[8] Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, White HD, ESC Scientific Document Group. Fourth universal definition of myocardial infarction (2018). Eur Heart J 2019;40:237_269. Investigators. Effect of definition on incidence and prognosis of type 2 myocardial infarction. J Am Coll Cardiol2017;70:1558_1568.

[9] Thygesen K, Alpert JS, Jaffe AS, *et al*. Third universal definition of myocardial infarction. J Am Coll Cardiol 2012;60: 1581-98.

[10] Murray CJ, Barber RM, Foreman KJ, *et al*. Global, regional, and national disability- adjusted life years (DALYs) for 306 diseases and injuries and healthy life expectancy (HALE) for 188 countries, 1990- 2013: quantifying the epidemiological transition. Lancet 2015; 386:2145-91.

[11] Libby P. Mechanisms of acute coro- nary syndromes and their implications for therapy. N Engl J Med 2013; 368:2004- 13.

[12] O'Gara PT, Kushner FG, Ascheim DD, *et al*. 2013 ACCF/AHA guideline for the management of ST-elevation myocardial infarction: areport of the American Col lege of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol 2013;61(4): e78-e140.

[13] Amsterdam EA, Wenger NK, Brindis RG, *et al*. 2014 AHA/ACC guideline for the management of patients with non-ST-ele- vation acute coronary syndromes: a report of the American College of Cardiology/ American Heart

Association Task Force on Practice Guidelines. J Am Coll Cardiol 2014;64(24):e139-e228.

[14] Savonitto S, Ardissino D, Granger CB, *et al*. Prognostic value of the admission electrocardiogram in acute coronary syndromes. JAMA 1999; 281:707-13.

[15] Welch RD, Zalenski RJ, Frederick PD, *et al*. Prognostic value of a normal or nonspecific initial electrocardiogram in acute myocardial infarction. JAMA 2001;286:1977- 84.

[16] Cannon CP, McCabe CH, Stone PH, *et al*. The electrocardiogrampredicts one-year outcome of patients with unstable angina andnon-Q wave myocardial infarction: results of the TIMI III Registry ECG Ancillary Study. Thrombolysis in Myocardial Ischemia. J Am Coll Cardiol1997; 30:133- 40.

[17] Westerhout CM, Fu Y, Lauer MS, *et al*. Short- and long-term risk stratification in acute coronary syndromes: the added value of quantitative ST-segment depression andmultiple biomarkers. JAmCollCardiol2006;48:939-47.

[18] Heeschen C, Hamm CW, Bruemmer J, Simoons ML. Predictive value of C reactive protein and troponin T in patients with unstable angina: acomparative analysis. CAPTURE Investigators. Chimeric c7E3 AntiPlatelet Therapy in Unstable angina Refractory to standard treatment trial. J Am Coll Cardiol2000;35:1535- 42.

[19] Heidenreich PA, Alloggiamento T, Melsop K, McDonald KM, Go AS, Hlatky MA. The prognostic value of troponin in patients with non-ST elevation acute coronary syndrome: a meta-analysis. J Am Coll Cardiol2001; 38:478-85.

[20] James SK, Lindbäck J, Tilly J, *et al*. Troponin-T and N-terminal pro-B-type natriuretic peptide predict mortality benefit from coronary revascularization in acute coronary syndromes: a GUSTO-IV substudy.J Am Coll Cardiol2006; 48:1146-54.

[21] Giannitsis E, Muller-Bardorff M, Lehrke S, *et al*. Admission troponin T level predicts clinical outcomes, TIMI flow, and myocardial tissue perfusion after primary percutaneous intervention for acute ST-segment elevation myocardial infarction. Circulation 2001;104: 630-5.

[22] Ohman EM, Armstrong PW, Christenson RH, *et al*. Cardiac troponin Tlevels for risk stratification in acute myocardial ischemia. GUSTO IIAInvestigators. N Engl J Med1996; 335:1333-41.

[23] Panteghini M, Cuccia C, Bonetti G, Giubbini R, Pagani F, Bonini E. Single-point cardiac troponin T at coronary care unit discharge after myocardial infarction correlates with infarct size and ejection fraction.Clin Chem 2002; 48:1432-6.

[24] de Lemos JA, Morrow DA, Bentley JH, *et al*. The prognostic value of B-type natriuretic peptide in patients with acute coronary syndromes. N Engl J Med 2001; 345:1014-21.

[25] James SK, Lindahl B, Siegbahn A, *et al*. N-terminal pro-brain natriuretic peptide and other risk markers for the separate prediction of mortality and subsequent myocardial infarction in patients with unstable coronary artery disease: a Global Utilization of Strategies to Open occluded arteries (GUSTO)-IV substudy. Circulation 2003; 108:275-81.

[26] Mega JL, Morrow DA, De Lemos JA, *et al*. B-type natriuretic peptide at presentation and prognosis in patients with ST-segment elevation myocardial infarction: an ENTIRE-TIMI-23 substudy. J Am Coll Cardiol2004;44:335-9.

[27] Sabatine MS, Morrow DA, Higgins LJ, *et al*. Complementary roles forbiomarkers of biomechanical strain ST2 and N-terminal prohormone B-type natriuretic peptide in patients with ST-elevation myocardial infarction. Circulation 2008; 117:1936 44.

[28] Anderson JL, Adams CD, Antman EM, *et al*. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non- ST-elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on PracticeGuidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients with Unstable Angina/Non-ST Elevation Myocardial Infarction). J Am Coll Cardiol 2007;50:e1-157.

[29] Hachamovitch R, Di Carli MF. Methods and limitations of assessing new noninvasive tests: Part II: outcomes-based validation andreliability assessment of noninvasive testing. Circulation 2008;117:2793-801.

[30] Antman EM, Anbe DT, Armstrong PW, *et al*. ACC/AHA guidelines for the management of patients with ST-elevation myocardial infarction–executive summary. A report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines (Writing Committee to revise the 1999 guidelines for the management of patients with acute myocardial infarction). J AmColl Cardiol2004; 44:671-719.

[31] Kumar V, Melhotra S, Ahuja Ret RC and Viash AK. Platelet and AcuteCoronary Syndrome. J Fam Med. 2016; 3(4): 1063.

[32] Jaya Manchanda, R M Potekar, Sharan Badiger, Abhishek Tiwari. Thestudy of platelet indices in acute coronary syndromes.Annals of Pathology and Laboratory Medicine, Vol. 02, No. 01, Jan-Mar 2015.

[33] Singhal G, Pathak V. The relationship between mean platelet volume and coronary collateral vessels in patients with acute coronary syndromes. Journal of the Practice of Cardiovascular Sciences. 2016;2(3):169.

[34] M M Khandekar, A S Khurana, S D Deshmukh, A L Kakrani, A D Katdare, A K Inamdar Platelet volume indices in patients with coronary artery disease and acute myocardial infarction: anIndian scenario J Clin Pathol. 2006 Feb; 59(2): 146-149.

[35] Biradar SB, Kashinakunti SV, Manjula R. Platelet volume indices in acute coronary syndrome- a case control study. Int J Adv Med 2016; 3:349-52.

[36] Dehghani MR, Taghipour-Sani L, Rezaei Y, Rostami R. Diagnostic importance of admission platelet volume indices in patients with acute chest pain suggesting acute coronary syndrome. Indian Heart J 2014;66(6): 622-8.

[37] Hendra TJ, Oswald GA, Yudkin JS. Increased mean platelet volume after acute myocardial infarction relates to diabetes and to cardiac failure. Diabetes Res Clin Pract 1988; 5: 63-69.

[38] Boos CJ, Balakrishnan B, Lip GY. The effects of coronary artery disease severity on time-dependent changes in platelet activation indices in stored whole blood. J Thromb Thrombolysis 2008 ;25(2): 135-40.

[39] Cameron HA, Philips R, Ibbotson RM, Carson PHM. Platelet size in myocardial infarction. Br Med J 1983;287: 449-51.

[40] Martin JF, Plumb J, Kilbey RS, Kishk YT Changes in volume and density of platelets in myocardial infarction. Br Med J 1983;287: 456- 9.

[41] Kumar V, Melhotra S, Ahuja Ret RC and Viash AK. Platelet and AcuteCoronary Syndrome. J Fam Med. 2016; 3(4): 1063.

[42] Jaya Manchanda, R M Potekar, Sharan Badiger, Abhishek Tiwari. The study of platelet indices in acute coronary syndromes.Annals of Pathology and Laboratory Medicine, Vol. 02, No. 01, Jan-Mar 2015.

[43] Assiri AS, Jamil AM, Mahfouz AA, Mahmoud ZS, Ghallab M. Diagnostic importance of platelet parameters in patients with acute coronary syndrome admitted to a tertiary care hospital in southwest region, Saudi Arabia. J Saudi Heart Assoc 2012;24(1): 17-21.

[44] Pervin S, Islam SM, Ferdoushi S, Hossain M, Sultana T, Hoque MH, *et al*. Platelet distribution width is an early indicator of acute coronary syndrome. University Heart Journal. 2013;9(1):3-8.

[45] Nandwani S, Bhatnagar M. Study of Platelet volume Indices in Plateletof Acute Coronary Events. JIAG. 2011;7:22-4.

[46] Akula, S., Krishna.K, V., J, R., Srinivas, B. and Damera, S. (2017). A Study of Platelet Indices in Acute Myocardial Infarction: AnObservational Study. IOSR Journal of Dental and Medical Sciences, 16(06), pp.10-13.

[47] Khode V, Sindhur J, Kanbur D, Ruikar K, Nallulwar S. Mean platelet volume and other platelet volume indices in patients with stable coronary artery disease and acute myocardial infarction: A case control study. J Cardiovasc Dis Res 2012;3:272-5.