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Original Research Article

Correlation of platelet indices with the spectrum of acute coronary syndrome and extent of coronary artery disease

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ABSTRACT

Introduction: Platelets have a major role in acute coronary syndrome and their activation is a hallmark of this. Troponin I, Troponin T and Creatine Kinase enzymes are not enough sensitive at an early stage of acute coronary syndrome. Platelet indices can be detected earlier, inexpensive, widely available and easily recordable in most clinical laboratories, thus could be a better marker in these patients. The primary objective of this study is to determine the correlation between platelet indices and spectrums of acute coronary syndrome or the number of vessels involved.

Material and Methods: This is a prospective observation study conducted in a tertiary care teaching hospital of eastern India over a period of six months where 125 patients were non-randomly selected with a diagnosis of acute coronary syndrome who underwent coronary angiogram and reports correlated with platelet parameters.

Results: A total of 100 patients were finally evaluated. Only platelet large cell ratio and platelet-crit were significantly higher in ST elevated myocardial infarction group compared to the unstable angina group. Across all the spectrums of acute coronary syndrome or extent of coronary artery disease there was a strong positive correlation within platelet distribution width, mean platelet volume and platelet large cell ratio, which was also very significant. Similarly total platelet count had a strong positive and very significant correlation with platelet-crit in the above groups.

Conclusion: Increase platelet large cell ratio and platelet-crit seems to be independent risk factors for development of ST elevated myocardial infarct than unstable angina. Thus they could serve as simple but important early biomarkers for predicting development of ST elevated myocardial infarction compared to unstable angina in acute coronary syndrome patients.

1. Introduction

Platelets have a major role in acute coronary syndrome (ACS) and their activation is a hallmark of this.[1] Platelets have also been implicated in the pathogenesis of cardio-vascular disorders including atherosclerosis and its complications, such as acute myocardial infarction (AMI), unstable angina (UA) and sudden cardiac death.[2] They play a crucial role in thrombus formation after rupture of the atherosclerotic plaque. There will be increased release of larger platelets with denser granules that are highly active.[3] Though Trop I, Trop T and Creatine kinase enzymes are more sensitive and specific biomarkers of myocardial damage, they still are not enough sensitive at an early stage of ACS, remaining undetectable in about 40 – 60% of patients.[4] Platelet indices can be detected earlier, relatively in expensive, widely available and also easily recordable in most clinical laboratories. Hence platelet parameters can be better used as markers for possible benefitting in timely intervention in the emergency department by improving risk

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stratification.[5]

The primary objective of this prospective observational study is to determine the correlation between platelet indices and the spectrum of ACS and to analyze if there exists a statistically significant difference between these indices and the numbers of vessels involved in patients admitted to the Cardiology ward and undergoing coronary angiogram.[6,7]

2. Materials and Methods

This is a prospective observational study conducted on a tertiary care teaching hospital of eastern India over a period of six months from Jan 2018 to July 2018. 125 patients selected non-randomly with a diagnosis of ACS admitted to the Cardiology department who fulfilled the inclusion and exclusion criteria were enrolled for this study.

25 no of patients were excluded thereafter due to insufficient and unreliable data or withdrawal from voluntary consent.

2.1. Inclusion criteria

Patients > 18 years of age presenting with pain chest consistent with ACS with any of the following feature were added into the study population.

1. Electrocardiographic (ECG Changes
   (a) ST elevation
   (b) ST depression
   (c) T inversion.
2. Trop T/Trop I /Creatine kinase MB isoform (CKMB elevation

2.2. Exclusion criteria

1. Patients already on antiplatelet/anticoagulant therapy
2. Patients with bleeding or clotting disorders.
3. Patients with blood/platelet product transfusion within last 3 months.
4. Primary platelet disorders, aplastic anemia.
5. Pregnancy, sepsis
6. Cancer, chronic kidney disease (CKD) with creatine clearance < 60 ml/min/m2body surface area (BSA).
7. Advanced liver diseases.
8. Patient on drugs that decreases cell count-Hydroxyurea, anti-neoplastic drugs.
9. Severe left ventricular (LV) systolic dysfunction with ejection fraction (EF) <30%.

All the study participants after giving there informed consent were subjected to focus history taking and clinical examination to obtain information related to demographic profile, risk factor, presenting symptoms, vital recordings and baseline 12 lead ECG. Information documented in a predesigned data sheet.

For measuring platelet indices blood samples were taken at the time of admission before starting any specific treatment. 2 ml of blood taken on the Ethylene diamine tetra acetic acid (EDTA) vacutainers obtained via antecubital venous access, examined within 30 minutes (6) by a fully automatic bidirectional hematology analyzer (SYSMEX XN 100) on flow cytometry principle for complete blood count. Platelet parameters like total platelet count (TPC), mean platelet volume(MPV), platelet distribution width(PDW), platelet large cell ratio(P-LCR) and platelet-crit (PCT) were noted. Other regular blood investigations were also performed.

Selective coronary angiogram was performed by femoral or radial approach by expert cardiologist. Angiogram results were interpreted by two different cardiologists according to quantitative coronary angiogram (QCA) method. Diameter stenosis > 50% in epicardial coronary arteries was accepted as significant. Left main stenosis of more than 30% was included in triple vessel disease (TVD). In patients with typical pain chest of > 20 minutes duration ST elevated myocardial infarct (STEMI) was defined as > 2 mm ST segment elevation at the 'J' point in at least 2 consecutive ECG leads from V1 to V3 or > 1 mm elevation in other leads. Non ST elevated myocardial infarct (NSTEMI) was defined as any ECG changes other than STEMI and/or typical pain chest with positive cardiac biomarkers more than two fold of upper limit of normal values. Unstable angina defined as typical pain chest and/or any of the ECG changes with non-diagnostic cardiac biomarkers.

2.3. Statistical analysis

Statistical analysis was done using R version 3.6.3 for calculating mean and standard deviation (SD) of the continuous variables. Comparison of the data distribution between groups was done using Mann Whitney Wilcoxon test. P value of less than or equal to 0.05 was considered significant. Pearson's correlation between the platelet indices was obtained using the Cor.test. Correlation coefficient of more than 0.7 was considered strong and less than 0.5 was considered weak relation.

3. Results

A total of 100 ACS patients were evaluated comprising of 78 STEMI patients, 18 NSTEMI patients and 04 UA patients. The age range of the patients was 27-75 years. The male patients compromised 87.4% of the study population. The base line characteristics of the study patients are summarized in Table 1.

Following coronary angiography the extent of coronary artery disease as per the number of vessels involved significantly by QCA method was as follows in Table 2.

All the Platelet parameters were found to be the highest in STEMI, followed by NSTEMI and the lowest

Table 1: The base line characteristics of the study group

Parameters	Values
Age range	27-75 years
Male Population	87.4%
Female Population	12.6%
Hypertension	36%
Diabetes mellitus	40.8%
Smoking	28.8%
Body mass Index	24 +3.4 (mean + SD)
Ejection fraction	55.34 + 11.3%(mean + SD)
LDL(low density lipoprotein)	100 to 8 + 34.2 mg%(mean + SD)
HDL(high density lipoprotein)	40.9 + 8.9mg%(mean + SD)
TG(triglyceride)	160.2 + 92.3mg%(mean + SD)
STEMI	78%
NSTEMI	18%
UA	4%

in UA patients, but the values of P-LCR and PCT were significantly increased in STEMI only when compared to UA patients (P=0.05, P=0.03) Table 3.

Table 2: Extent of Coronary Artery Disease (CAD)

Number of Vessels involved	n (%)
Single vessel disease (SVD)	52 (52)
Double vessel disease (DVD)	28 (28)
Triple vessel disease (TVD)	20 (20)

Linear regression analysis performed to determine the Pearson's correlation between one platelet parameter with others. Statistically very significant and strong positive correlation was observed amongst PDW, PLCR and MPV (r=0.9) (Tables 4, 5 and 6). Similarly TPC and PCT are very significantly related to each other with a strong positive correlation (r=0.9) (Tables 7 and 8). These relations are seen across the entire spectrum of ACS.

There was no significant difference of the platelet indices according to the number of vessels significantly involved by QCA method (Table 9).

PDW, PLCR and MPV are very significantly related to each other with a strong positive association (r= 0.8 to 0.9) (Tables 10, 11 and 12). Similarly TPC and PCT are also very significantly related to each other with a strong positive association(r=0.9) (Tables 13 and 14). Both the relations are found irrespective of the extent of CAD (whether SVD, DVD or TVD).

4. Discussion

Platelet activation favours thrombus formation and coronary artery occlusion thus playing a key pathogenic role in AMI. Platelets are also heterogeneous in terms of size, density and activity. Larger hyperactive platelets may play an important role in thrombus formation, resulting in acute thrombotic events.[8] Release of larger platelets from bone marrow could follow decrease of TPC due to their consumption at the site of thrombosis.[9] Thus these markers could maintain their strength and predictive value in ACS patients. Automated cell counters in hospital laboratories have made platelet indices available routinely and effortlessly as a byproduct, which can be added as a cost effective tool in diagnosis and prognosis of ACS spectrum and CAD extent.

In the present study we measured the platelet parameters in patients suffering from ACS with age range of 27-75 years comprising of 87.4% male and 12.6% females. In our study apart from P-LCR & PCT other platelet indices did not show a significant difference amongst various spectrum of ACS. This is comparable to the study, conducted by Gargi G et al.[10] The value of P-LCR was significantly increased in STEMI patients compared to UA patients. This is also comparable to the results of the study by Ranjith MP et al.[11] wherein PLCR was significantly higher in patients of ACS, compared to control population. This could be because P-LCR is another index of platelet volume. The study conducted by Sermin et al showed that MPV value of STEMI patients was slightly higher than NSTEMI patients but without reaching statistical significance.[12] Our study similarly showed MPV to be more in STEMI than NSTEMI and least in UA without any statistically significant difference. Our study also showed that TPC is highest in STEMI, followed by NSTEMI and least in UA, without intergroup statistical significance. Similar result was also observed by Dehghani et al,[13] where platelet count was 2.8% more in MI compared to UA but P was 0.16. So also in that study MI patients had significantly higher PLCR than UA, as seen in the present study.

We found statistically significant weak negative correlation within TPC and platelet volume indices, which was also seen by Bhawani YA et al.[4] Pearson correlation analysis done to determine the relation of the TPC with other platelet indices showed that it is having a negative but significant correlation with MPV and PDW in STEMI patients in the study by Reddy SK et al.[14] Similar to that our study shows that TPC is having significant negative correlation with all platelet volume indices in STEMI patients. But in TVD group this is restricted to PDW and PLCR but not MPV. Increased MPV and PDW are known to be associated with increased morbidity, mortality and recurrent MI.[15,16] Thus they could be simple and reliable biomarkers to predict significant and severe coronary events. In the study by Dehghani et al Pearson correlation analysis done had shown that significant negative correlation exist between TPC and MPV or PLCR but not PDW (0.07) in MI patients. But here this relation is applicable to all volume indices in STEMI patients.

MPV value of more than 9.0 fL is usually defined as high. Though in patients of ACS, MPV is found to be high across

Table 3: Comparison of platelet indices between different spectrums of ACS

Platelet Indices	STEMI (I)n(78)mean + SD	NSTEMI (II)n(18)mean + SD	UA (III)n(4)mean + SD	P Value(d) A	b	C
MPV(fL)	11.74+1.76	11.29+2.70	10.5+4.44	0.76	0.78	1.0
PDW(fL)	15.02+3.58	14.56+4.38	14.24+5.92	0.74	0.93	0.93
TPC (x$10^3/\mu$L)	264.69+92.59	231+78.87	192.96+82.83	0.23	0.07	0.32
PLCR(%)	38.82+10.17	37.92+12.78	28.45+12.03	0.98	0.05^d	0.08
PCT(%)	0.31+0.10	0.26+0.08	0.21+0.09	0.06	0.03^d	0.27

a - P value within group I and II
b- P Value within group I and III
c- P Value within group II and III
d- P value <0.05 was considered statistically significant.

Table 4: Pearson Correlation (r) between PDW and other platelet indices in various spectrums of ACS

PDW Vs.	STEMI		NSTEMI	UA
TPC	r value	-0.45^f	-0.24	0.09
	p value	3.48E-05^d	0.31	0.87
MPV	r value	0.91^e	0.91^e	0.96^e
	p value	2.20E-16^d	3.25E-08^d	0.003^d
PLCR	r value	0.91^e	0.97^e	0.94^e
	p value	2.20E-16^d	1.03E-12^d	0.004^d
PCT	r value	-0.32^f	0.06	0.25
	p value	4.42E-03^d	8.11E-01	0.63

d – P value of < 0.05 was considered statistically significant.
e – Pearson correlation co-efficient of >0.7 indicates strong relation.
f- Pearson correlation co-efficient of <0.5 indicates weak relation.

Table 5: Pearson Correlation (r) between PLCR and other platelet indices in various spectrums of ACS

PLCR Vs.	STEMI		NSTEMI	UA
TPC	r value	-0.39^f	-0.35	0.39
	p value	3.07E-04^d	0.13	0.44
MPV	r value	0.86^e	0.87^e	0.99^e
	p value	2.20E-16^d	7.62E-07^d	0.000^d
PDW	r value	0.91^e	0.97^e	0.94^e
	p value	2.20E-16^d	1.03E-12^d	0.005^d
PCT	r value	-0.24^f	-0.04	0.55
	p value	3.18E-02^d	8.68E-01	0.26

d – P value of < 0.05 was considered statistically significant.
e – Pearson correlation co-efficient of >0.7 indicates strong relation.
f- Pearson correlation co-efficient of < 0.5 indicates weak relation.

Table 6: Pearson Correlation (r) between MPV and other platelet indices in various spectrums of ACS

MPV Vs.	STEMI		NSTEMI	UA
TPC	r value	-0.27^f	0.06	0.38
	p value	0.02^d	0.80	0.46
PDW	r value	0.91^e	0.91^e	0.96^e
	p value	2.20E-16^d	3.25E-08^d	0.003^d
PLCR	r value	0.86^e	0.87^e	0.99^e
	p value	2.20E-16^d	7.62E-07^d	0.000^d
PCT	r value	-0.14	0.33	0.52
	p value	2.08E-01	1.50E-01	0.29

d – P value of < 0.05 was considered statistically significant
e – Pearson correlation co-efficient of >0.7 indicate strong relation
f- Pearson correlation co-efficient of <0.5 indicates weak relation

Table 7: Pearson Correlation (r) between TPC and other platelet indices in various spectrums of ACS

TPC Vs.	STEMI		NSTEMI	UA
MPV	r value	-0.27[f]	-0.06	0.38
	p value	0.02[d]	0.80	0.46
PDW	r value	-0.45[f]	-0.24	0.09
	p value	3.48E-05[d]	0.31	0.87
PLCR	r value	-0.39[f]	-0.35	0.39
	p value	0.000[d]	0.13	0.44
PCT	r value	-0.92[e]	0.94[e]	0.98[e]
	p value	2.20E-16[d]	4.84E-10[d]	0.000[d]

d – P value of < 0.05 was considered statistically significant
e – Pearson correlation co-efficient of >0.7 indicates strong relation
f- Pearson correlation co-efficient of <0.5 indicates weak relation

Table 8: Pearson Correlation (r) between PCT and other platelet indices across various spectrums of ACS

PCT Vs.	STEMI		NSTEMI	UA
TPC	r value	0.87[e]	0.97[e]	0.91[e]
	p value	1.74E-07[d]	2.20E-16[d]	2.20E-16[d]
MPV	r value	0.21	0.05	-0.04
	p value	3.52E-01	8.00E-01	0.78
PDW	r value	-0.04	-0.18	-0.23
	p value	8.46E-01	3.46E-01	0.09
PLCR	r value	-0.000	-0.16	-0.14
	p value	9.99E-01	3.87E-01	0.31

d – P value of < 0.05 was considered statistically significant
e – Pearson correlation co-efficient of >0.7 indicates strong relation

Table 9: Comparison of platelet indices with different extent of CAD

Platelet Indices	SVD (I)n(52)mean + SD	DVD (II)n(28)mean + SD	TVD (III)n(20)mean + SD	P Value(d) a	b	C
MPV(fL)	11.13+2.5	11.89+2.44	11.59+1.93	0.10	0.37	0.25
PDW(fL)	14.17+4.02	15.63+4.31	14.78+3.58	0.17	0.46	0.39
TPC (x$10^3/\mu$L)	238.90+81.06	243.31+93.94	260.59+87.30	0.96	0.27	0.31
PLCR(%)	35.36+10.13	41.06+12.08	37.60+10.52	0.06	0.40	0.15
PCT(%)	0.27+0.09	0.29+0.10	0.30+0.10	0.95	0.33	0.42

a - P value within group I and II
b-P Value within group I and III
c-P Value within group II and III
d- P value <0.05 was considered statistically significant.

Table 10: Pearson Correlation (r) between PDW and other platelet indices across various extent of CADd – P value of <0.05 was considered statistically significant

PDW Vs.	SVD		DVD	TVD
TPC	r value	-0.15	-0.35	-0.43[f]
	p value	0.51	0.06	0.001[d]
MPV	r value	0.89[e]	0.91[e]	0.92[e]
	p value	4.48E-08[d]	1.78E-12[d]	2.20E-16[d]
PLCR	r value	0.92[e]	0.94[e]	0.90[e]
	p value	1.67E-09[d]	6.77E-15[d]	2.20E-16[d]
PCT	r value	-0.04	-0.18	-0.23
	p value	8.46E-01	3.46E-01	8.74E-02

e – Pearson correlation co-efficient of >0.7 indicates strong relation
f- Pearson correlation co-efficient of <0.5 indicates weak relation

Table 11: Pearson Correlation (r) between PLCR and other platelet indices across various extent of CAD

PLCR Vs.	SVD		DVD	TVD
TPC	r value	-0.13	0.35	-0.38[f]
	p value	0.57	0.06	0.004[d]
MPV	r value	0.84[e]	0.87[e]	0.85[e]
	p value	1.17E-06[d]	7.21E-10[d]	4.93E-16[d]
PDW	r value	0.92[e]	0.94[e]	0.90[e]
	p value	1.67E-09[d]	6.77E-15[d]	2.20E-16[d]
PCT	r value	-0.0003	-0.16	-0.14
	p value	9.99E-01	3.87E-01	3.11E-01

d – P value of < 0.05 was considered statistically significant.
e – Pearson correlation co-efficient of >0.7 indicates strong relation.
f- Pearson correlation co-efficient of <0.5 indicates weak relation.

Table 12: Pearson Correlation(r) between MPV and other platelet indices across various extent of CAD

MPV Vs.	SVD		DVD	TVD
TPC	r value	0.16	-0.11	-0.21
	p value	0.49	0.57	0.12
PDW	r value	0.89[e]	0.91[e]	0.92[e]
	p value	4.48E-08[d]	1.78E-12[d]	2.20E-16[d]
PLCR	r value	0.84[e]	0.87[e]	0.85[e]
	p value	1.17E-06[d]	7.21E-10[d]	4.93E-16[d]
PCT	r value	0.21	0.05	-0.04
	p value	3.52E-01	8.00E-01	7.81E-01

d – P value of < 0.05 was considered statistically significant
e – Pearson correlation co-efficient of >0.7 indicates strong relation

Table 13: Pearson Correlation (r) between PCT and other platelet indices across various extent of CAD

PCT Vs.	SVD		DVD	TVD
TPC	r value	0.87[e]	0.97[e]	0.91[e]
	p value	1.74E-07[d]	2.20E-16[d]	2.20E-16[d]
MPV	r value	0.21	0.05	-0.04
	p value	3.52E-01	8.00E-01	7.81E-01
PDW	r value	-0.04	-0.18	-0.23
	p value	8.46E-01	3.46E-01	8.74E-02
PLCR	r value	-0.0003	-0.16	-0.14
	p value	9.99E-01	3.87E-01	3.11E-01

d – P value of < 0.05 was considered statistically significant.
e – Pearson correlation co-efficient of >0.7 indicates strong relation.

Table 14: Pearson Correlation (r) between TPC and other platelet indices across various extent of CAD

TPC Vs.	SVD		DVD	TVD
MPV	r value	0.16	-0.11	-0.21
	p value	0.49	0.57	0.12
PDW	r value	-0.15	-0.35	-0.43[f]
	p value	5.13E-01	0.06	0.001[d]
PLCR	r value	-0.13	-0.35	-0.38[f]
	p value	0.57	0.06	0.004[d]
PCT	r value	0.87[e]	0.97[e]	0.91[e]
	p value	1.74E-07[d]	2.20E-16[d]	2.20E-16[d]

d – P value of < 0.05 was considered statistically significant.
e – Pearson correlation co-efficient of >0.7 indicates strong relation.
f- Pearson correlation co-efficient of <0.5 indicates weak relation.

the spectrums, there is no significant relation of severity of CAD with their MPV values. This was also seen by Reddy Sk et al.[14] In another large scale study by De Luca G et al[17] there was no correlation between MPV or PDW and the extent of CAD, according to coronary angiogram either in ACS or elective cases. Similarly in our study none of the volume indices is related to extent of CAD in angiography of ACS patients.

In the earlier largest study so far PWD was inversely related to TPC in a significant manner (P<0.001). Our study also showed significant negative correlation between PDW and TPC (P=0.001) but only in patients of TVD. But in STEMI patients this negative correlation was found for both TPC (P=3.48 E-05) and PCT (P=4.42 E-03).

5. Conclusion

Irrespective of the spectrum of ACS or extent of CAD, platelet volume indices are very significantly associated with each other in a strong positive way. Similarly TPC and PCT are having such aforementioned relation with each other. However though the study showed that PDW and MPV may not be related to the spectrum of ACS or to the CAD extent, increased value of PLCR and PCT seems to be independent risk factors for development of STEMI than UA. In addition in ACS patients TPC is having a mild negative but significant correlation with all platelet volume indices only in STEMI patients, but it has similar relation with only PDW and PLCR in TVD patients.

There simple, reliable, easy to perform, noninvasive and economic method may predict the risk of STEMI in ACS presentation, thus could serve as important tools for risk stratification in them.

But conflicting results of various platelet indices in difference studies emphasizes that further large scale trials should be conducted in future to include those simple platelet parameters in triaging of ACS patient management.

6. Limitations

This study had a smaller sample size and long term follow up was not done for prognostic evaluation of those parameters. Risk factors HT, DM, smoking and drugs like Atorvastasin,[18] Insulin,[19] nonsteroidal anti-inflammatory drugs (NSAIDS) and Caffeine,[20] could act as confounding factors. Furthermore IVUS could have provided more accurate information on the severity of CAD and plaque burden which could not be done in this study. Conventional cardiac biomarkers could also have been compared with platelet indices for better understanding of the process.

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9. Conflict of Interest

None.

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