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A COMPARATIVE STUDY OF PLATELET INDICES IN CORONARY ARTERY DISEASES

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ABSTRACT

INTRODUCTION: With urbanization and an increase in the sedentary lifestyle, prevalence of CAD is looming large as the new epidemic afflicting. Altered platelet morphology and functions have been linked with the formation and propagation of thrombotic events. Platelet indices (Platelet count- PC, Mean platelet volume - MPV, Platelet distribution width - PDW, Platelet large cell ratio - P-LCR) are determinants of platelet function.

AIMS AND OBJECTIVES: 1. To study the platelet indices (PC, MPV, PDW, P-LCR) in coronary artery diseases

2. To compare the platelet indices in patients with myocardial infarction, stable CAD and control population.

MATERIAL & METHODS: A comparative and prospective study was conducted on 100 patients each of MI & stable CAD on antiplatelet therapy and compared with the healthy individuals as controls. Platelet indices were measured using an Automated Blood Counter SYSMEX

XN-1000. Troponin T and CK-MB levels were collected from clinical data in MI cases.

RESULTS: Platelet indices were significantly higher in MI patients in comparison to the stable CAD and control groups. Stable CAD patients also showed significantly higher platelet indices in comparison to the control groups. (p value <0.001) xiii

CONCLUSION: The present study showed a significantly higher MPV, PDW and P-LCR in patients with CAD in comparison to the control group. Among the CAD patients, patients with MI had significantly higher platelet indices than stable CAD patients.

Hence platelet indices can be used as simple and cost effective predictive parameters to predict CAD. Their use in a risk stratification system to predict MI as well as in response to the intervention are worthy of consideration.

KEYWORDS : Coronary artery diseases, Platelet indices, Mean platelet volume, Platelet distribution width, platelet large cell ratio.

INTRODUCTION

Coronary artery disease (CAD) is the leading cause of mortality and morbidity in the world and acute coronary syndromes (ACS), which encompass the unstable angina (UA), non-ST-segment elevation myocardial infarction (NSTEMI) and ST segment elevation myocardial infarction (STEMI). The prevalence of CAD and the incidence of ACS also are very high among Indians.1-5

Altered platelet morphology and functions have been linked with the pathogenesis of the CAD. Hyperactivity of platelets have an important role in the initiation of atherosclerotic lesions and coronary thrombogenesis. Larger platelets are more active enzymatically and metabolically and have a higher thrombotic ability as compared to the small sized platelets[6,7]. Platelet indices (PC, MV, PDW, P-LCR) are the determinants of platelet function, among which, an increased MPV and PDW have been found to be important contributory factors causing thromboembolism[6]Patients with larger platelets can easily be identified during routine hematological analysis and could possibly benefit from the preventive/therapeutic treatment. This study was aimed at finding a link between CAD and platelet indices.

OBJECTIVES OF THE STUDY

1. To study the Platelet indices (PC, MPV, PDW, P-LCR) in coronary artery diseases.
2. To compare the Platelet indices in patients with myocardial infarction, stable CAD and the control population.

MATERIALS AND METHODS:

Source of data:

This is an analytical study undertaken in a tertiary health care centre for a period of 2 years. Study population was divided into three groups as follows:

Group 1: 100 patients with Myocardial infarction not on antiplatelet treatment

Group 2: 100 patients with stable CAD on antiplatelet treatment

Group 3: 100 age and sex matched healthy individuals

INCLUSION CRITERIA:

Diagnosed cases of coronary artery diseases.

Exclusion criteria:

Patients with a history of renal disease, a history of past coronary intervention or coronary arterial bypass grafting, a history of inflammatory rheumatic disease, a history of chronic obstructive pulmonary disease and taking oral anticoagulants were excluded. Patients with Myocardial infarction pretreated or loaded with antiplatelet drugs were excluded.

Method of collection of data:

a. Data collection was done in a predesigned proforma.
b. Venous blood samples were collected in a vacutainer containing di-potassium
E. DTA and processed within 2 hours of sample collection.
c. Platelet indices (PC, MPV, PDW, P-LCR) were measured in cases and control groups using an Automatic Blood Counter (SYSMEX, XN-1000)
d. Serum Troponin T and CK-MB levels of MI patients were collected from the clinical data.

STATISTICAL METHODS

Descriptive statistics such as numbers and percentages were used to describe categorical variables. Mean and standard deviations were used to describe continuous variables like platelet count, MPV, PDW and P-LCR. Independent sample t-test was applied to find out the significant difference in platelet count, MPV, PDW and P-LCR between the cases and controls. Pearson's correlation was used to analyze association between different variables. Statistical significance was determined at 5% level of significance (i.e. < 0.05 is significant). Statistical analysis was done using Statistical package for social sciences (SPSS version 22) software. Microsoft word and Excel were used to generate graphs, tables and etc.

RESULTS

The present study included 100 diagnosed cases each of myocardial infarction, stable CAD

over a period of two years. Platelet indices and serum markers were recorded in a proforma. The details were then transcribed into master chart were then analyzed.

Age of patients with MI ranged from 32 years to 85 years with a mean age of 64.2 ± 11.52 years. Age of the patients with stable CAD ranged from 25 years to 81 years with a mean age of 61.7 ± 11.88 years. Age of the controls ranged from 52 years to 89 years with a mean age of 67.6 ± 10.71 years. Out of 100 patients with MI, number of males and females were 64 and 36, respectively. Out of 100 patients with stable CAD, number of males and females were 67 and 33 respectively. Controls had 59 males and 41 females. Out of 100 patients with MI, 78 were ST elevation MI (STEMI) and 22 were Non-ST elevation MI (NSTEMI) Mean platelet count of the patients with MI, stable CAD and controls were 2.78 ± 0.77, 3.32 ±0.86 and 2.9 ± 0.65 (106 / L), respectively with no significant difference (p value – 0.41). The mean MPV of the patients with MI, stable CAD and controls were 10.5 ± 0.76, 10.2 ± 0.87, 9.4 ± 0.7 fL, respectively. There was a significant difference between the MPV of these groups (p <0.001) with highest mean MPV being of the patients with MI.

The mean PDW of the patients with MI, stable CAD and controls were 12.1±1.68, 11.8±2.22 and 9.8±1.4 fL, respectively. The PDW was found to be significantly higher in patients with MI compared with the patients with stable CAD and control group. (p < 0.0001).

The mean P-LCR in the patients with MI, stable CAD and controls were 28.3±5.87, 26.6±7.22 and 20.1±5.84 %, respectively. The P-LCR was significantly higher in MI group in comparison to the patients with stable CAD and control group (p < 0.0001).

There was no significant correlation of platelet indices with serum Troponin T and CK-MB levels. (p > 0.05)

DISCUSSION

CAD is the leading cause of mortality and morbidity in India.4Altered platelet morphology and functions have been linked with the pathogenesis of CAD. When platelets come in contact with ruptured plaque, they become hyperactive and larger in size. These larger platelets are more active enzymatically and metabolically and have a higher thrombotic ability as compared to the small sized platelets because of a higher production of thromboxane A2 [6,7] In addition to the generation of thromboxane A2, activation of platelets leads to conformational change in GP IIb/IIIa receptor. This receptor develops a high affinity for fibrinogen. Since fibrinogen is a multivalent molecule, it can bind to two different platelets simultaneously, resulting in platelet cross-linking and aggregation.[8,9] This increase in platelet consumption at the site of the atherosclerotic plaque causes larger platelets to be released from the bone marrow[10,11] Presently therapeutic implications regarding platelets are restricted to their numbers only. These larger platelets can easily be identified during routine hematological analysis by platelet volume indices (PC, MPV, PDW, P-LCR)[6] The present study was conducted to determine the relationship of platelet indices with CAD and to compare the platelet indices in myocardial infarction, stable CAD and control population. 100 cases of each MI, stable CAD cases and age and sex matched controls were studied in the present

PLATELET COUNT AND CAD

Mean platelet count was observed to be the lowest in patients with MI in comparison to the patients with stable CAD. This may be due to the immediate increased consumption of platelets at the site of plaque rupture.[12] However, there was no significant difference among the mean platelet count of all the 3 groups. This finding was supported by Amraotkar et al[13] and Khode et al[14] ,but it was in contrast to the studies conducted by Khandekar et al[11] and Ranjith et al[12].study

MPV AND CAD

Mean MPV was significantly higher in patients with CAD than in controls. A significant difference was noted in the mean MPV of patients with MI and stable CAD (10.45 ± 0.76 and 10.21 ± 0.87 fL, respectively). This was in concordance with the studies by Amraotkar et al[13], Khode et al[14] and Ranjith et al[12]. A rise in MPV seen during MI may be due to increased platelet reactivity, which in turn increases the platelet surface expression of IIb/IIIa receptors and P selectin proteins. According to our study, MPV was higher in the patients with MI and stable CAD (SCAD) compared with the normal population. A possible explanation for an increased platelet volume and MPV is the increased platelet activity and activation of the coagulation cascade. (Table 1) Table 1: Comparision of MPV in MI and Stable coronary artery disease (SCAD) with other studies.

Study	Mean MPV (fL) in MI	Mean MPV (fL) in SCAD	P value
Khandekar et al[11]	10.43 ± 1.03	9.37 ± 0.99	< 0.001
Khode et al[14]	9.65 ± 0.9	9.38 ± 0.8	0.025
Ranjith et al[12]	10.97 ± 0.58	10.03 ± 0.23	< 0.001
Sharma et al[10]	10.29 ± 1.12	9.19 ± 0.62	< 0.001
Amraotkar et al[13]	9.18 ± 1.21	8.13 ± 0.66	0.003
Present study	**10.45 ± 0.76**	**10.21 ± 0.87**	**0.000**

PDW AND CAD

A significant difference was noted between mean PDW of patients with CAD and controls. The mean PDW of the patients with MI and stable CAD were 12.06 ± 1.69 and 11.77 ± 2.22 fL, respectively. A highly significant difference was also noted in the PDW among these groups. This result was in concordance with studies conducted by Khandekar et al, [11] Ranjith et al[12] and Sharma et al. [10] However, Khode et al[14] did not find any significant difference in PDW among these groups. It is argued that this platelet volume distribution provides a signature for a prethrombotic state in IHD.

Hence, considering that PDW is an index of platelet heterogeneity, this may explain the above witnessed increase in MI patients.[12] (Table 2)

Table 2: Comparision of PDW in MI and Stable coronary artery disease (SCAD) with other studies.

Study	Mean PDW(fl) in MI	Mean PDW(fl) in SCAD	P value
Khandekar et al[11]	13.19 ± 2.34	11.35 ± 1.95	< 0.001
Khode et al[14]	10.84 ± 2.2	10.65 ± 1.7	0.376
Ranjith et al[12]	14.63 ± 0.64	12.43 ± 0.62	< 0.001
Sharma et al[10]	15.11 ± 0.88	13.25 ± 0.44	< 0.001
Present study	**12.06 ± 1.69**	**11.77 ± 2.22**	**0.000**

P-LCR AND CAD

There was a significant difference between mean P-LCR of the patients with CAD and controls. The mean P-LCR of the patients with MI and stable CAD were 28.32 ± 5.87 and 26.59 ± 7.22 %, respectively with a significant difference. This was further supported by Khandekar et al[11] and Ranjith et al.[12] However, Khode et al[14] did not find any significant difference in P-LCR among these groups. (Table 3)

Table 3: Comparision of P-LCR in MI and Stable coronary artery disease (SCAD) with other studies.

Study	Mean P-LCR(%) in MI	Mean P-LCR(%) in SCAD	P value
Khandekar et al[11]	29.4 ± 7.38	22.55 ± 6.65	< 0.001
Khode et al[14]	21.58 ± 6	20.92 ± 6.4	0.315
Ranjith et al[12]	32.23 ± 1.94	26.77 ± 1.08	< 0.001
Present study	**28.32 ± 5.87**	**26.59 ± 7.22**	**0.000**

CONCLUSION

We conclude that platelet indices, which are simple and cost effective predictive parameters of platelet activation, can be used in conjunction with other laboratory tests for patients admitted to the emergency department (particularly in remote centres and in centres where cardiac serum markers are not readily available) with

chest pain to predict the development of acute coronary events. This could help to avoid hospitalization and also misdiagnosis. Future studies including the use of platelet indices in a risk stratification system to predict MI as well as in response to intervention are worthy of consideration.

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