

Mean Platelet Volume and Other Platelet Volume Indices in Acute Myocardial Infarction (AMI) and Stable Coronary Artery Diseases (SCAD): A Hospital Based Prospective Observational Study

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ABSTRACT

Introduction: Myocardial infarction is the major cause of morbidity and mortality in industrialized countries. Platelet activation is a hallmark of acute coronary syndrome. An increased MPV, an indicator of larger and more reactive platelets, has been associated with myocardial damage. Study aimed to record the platelet volume indices in Acute Myocardial Infarction (AMI), unstable angina (UA), stable coronary artery diseases (SCAD) and compare them with age and sex matched controls and to attempt a clinicopathological correlation.

Material and methods: This was a comparative study of 286 subjects; 39 patients with AMI, 49 patients with SCAD and 198 were controls. MPV and other PVI were assessed by their venous samples.

Results: The mean platelet volume was significantly higher in patients with AMI (9.30 ± 0.91) as compared to SCAD in patients were (8.38 ± 0.85) and controls (7.71 ± 1.05).

Conclusion: Thus MPV is considered as a cost effective tool that may reflect an atherosclerothrombotic tendency in human body.

Keywords: Acute Myocardial Infarction, Coronary Artery Disease, Stable Coronary Artery Disease, Mean Platelet Volume, Platelet Volume Indices.

INTRODUCTION

In this industrial world, coronary artery disease (CAD) is emerging as the major break through affecting people world wide and Indian also. Myocardial infarction is the major cause of morbidity and mortality in industrialized countries.¹ Acute coronary syndrome (ACS) is a set of sign and symptoms caused by rupture of an arterial plaque. Which provokes platelet rich coronary thrombus formation. The thrombus leads to partial or complete coronary artery occlusion, which in turn, results in myocardial ischemia and various clinical manifestations ranging from unstable angina (UA) to acute myocardial infarction (AMI).²

Platelets are the blood cells with variable sizes and densities. Platelet activation is a hallmark of acute coronary syndrome.³ Platelets have been implicated in the pathogenesis of cardiovascular disorders, including atherosclerosis and its complications, such as AMI, UA and sudden cardiac death.⁴ It has been shown that platelets size, when measured as mean platelet volume (MPV), is marker of platelet function and is positively associated with indicators of platelets activity. An increased MPV, an indicator of larger and more reactive platelets, has been associated with myocardial damage in

ACS and has been found to be predictive of an unfavorable outcome among survivors of AMI.^{3,4}

Automated cell counters have made the platelets count (PC) and the platelets volume indices (PVI) – mean platelet volume (MPV), platelet distribution width (PDW) and platelet (PCT) – routinely available in most clinical laboratories.

Study aimed to record the platelet volume indices in Acute Myocardial Infarction (AMI), unstable angina (UA), stable coronary artery diseases (SCAD) and compare them with age and sex matched controls and to attempt a clinicopathological correlation.

MATERIAL AND METHODS

A prospective study was carried out on 286 patients over a period of 6 months from December 2012 to May 2013.

Total 286 subjects were studied in 3 groups:-

Group I A:- Patients admitted to the ICU with UA and for AMI

Group I B:- Patients with SCAD admitted for coronary angiography and coronary artery bypass grafting (CABG) procedure and patients came for follow up, having a previous ischemic event.

Group II: - Age and Sex matched normal healthy controls with a normal ECG.

In group I A patients, we collected blood samples within six hours of admission before administration of anticoagulants and antiplatelet drugs. Blood samples of group I B patients were collected on the day of admission. Group II subjects came for routine checkup. All blood samples were collected in vacutainers containing EDTA. Samples were analyzed within 2 hours of collection with HMX coulter system and all the platelets parameters, hemoglobin and total leukocyte count were noticed and analyzed.

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In group I A patients AMI was diagnosed based on the following criteria:- Detection of rise or fall in cardiac biomarker Trop-I or CKMB with at least one value above 99th percentile of upper limit together with evidence of myocardial ischemia based on at least one of the following-

1. Symptoms of Ischemia
2. ECG changes indicative of new ischemia
3. Development of pathological Q wave in the ECG
4. Imaging evidence of new loss of viable myocardium or a new regional wall motion abnormality.⁵

In Group I B were diagnosed based on the following criteria:
 - Patients with SCAD admitted for coronary angiography and coronary artery bypass grafting (CABG) procedure and patients came for follow up, having a previous ischemic event

STATISTICAL ANALYSIS

Results were presented as mean, SD or frequency as appropriate. Other than these two sample t test, one way ANOVA was used for stastical analysis of the data. for comparison of the three groups ANOVA was used and for the comparison of two groups t test was used. A p value of, 0.05 was considered statistically significant. For the analysis

of the data SPSS 20 was used.

RESULTS

During 6 months of study, total 286 individuals were studied under the three groups.

AMI was diagnosed in 39 patients(group 1a). 46 patients were under SCAD (group1b) and total 198 subjects were age and sex matched controls (group II). All hematological parameters were generated by Beckman coulter analyzer and analyzed stastically. Large platelets were also confirmed by peripheral blood smear.

The mean age (SD) of the CAD patients was 60.23(12.05) and healthy control was 53.10 (7.41).

No significant difference was observed in Hb value in three groups. The white blood cell count was significantly raised in AMI group compared with SCAD and control group (Table1).

Platelet volume indices –MPV and PDW were significantly raised in AMI compared with SCAD and control group (Table 1). Although platelet counts were with in normal range in all the three grps but there was a significant differenece (p value < 0.05) in three groups. There was no significant difference obseved in value of pct.

Sr. No.	Parameter	Group 1A, AMI N = 39	Group 1B, SCAD n = 49	Group 2, Controls n =198	F Test	P Value
1	Age (yrs)	61.72 ± 12.51	59.04 ± 11.67	53.10 ± 7.41	19.77	<0.0001
2	Hb (gm/dl)	12.65 ± 2.13	12.71 ± 1.15	12.89 ± 0.78	1.35	0.3230
3	WBC (10 ³ / μl)	13.24 ± 4.87	8.79 ± 3.57	6.15 ± 1.40	128.9	<0.0001
4	PLT (10 ⁵ / μl)	2.01 ± 0.72	2.39 ± 0.79	2.74 ± 0.95	12.95	<0.0001
5	MPV (fl)	9.30 ± 0.91	8.38 ± 0.85	7.71 ± 1.05	44.23	<0.0001
6	PDW (ratio)	16.45 ± 0.79	16.03 ± 2.46	15.63 ± 0.70	8.679	0.0002
7	PCT (%)	0.186 ± 0.647	0.207 ± 0.071	0.209 ± 0.077	1.431	0.2708

Table-1: Comparison of Hematological parameters in all the three groups

Sr. No.	Parameter	Group 1 CAD n = 88	Group 2, Controls n =198	T Test	P Value
1	Age (yrs)	60.23 ± 12.05	53.10 ± 7.41	6.126	<0.0001
2	Hb (gm/dl)	12.68 ± 1.65	12.89 ± 0.78	- 1.489	0.138
3	WBC (10 ³ / μl)	10.76 ± 4.72	6.15 ± 1.40	12.592	<0.0001
4	PLT (10 ⁵ / μl)	2.22 ± 0.78	2.74 ± 0.95	- 4.639	<0.0001
5	MPV (fl)	8.70 ± 0.99	7.71 ± 1.05	8.128	<0.0001
6	PDW (ratio)	16.22 ± 1.91	15.63 ± 0.70	3.806	<0.0001
7	PCT (%)	0.198 ± 0.068	0.209 ± 0.077	- 1.109	0.269

Table-2: Comparison of Hematological parameters in CAD and control groups

	Previous studies	AMI		CONTROLS		P value
		N	MPV	N	MPV	
1	O'Brien et al(1973) ⁶	23	8.10	36	7.01	<0.001
2	Cameron et al(1983) ⁷	100	9.07	200	8.32	<0.001
3	Martin et al(1983) ⁸	15	7.30	22	6.32	0.05
4	Martin et al(1991) ⁹	126	10.09	1590	9.72	<0.001
5	Smyth et al(1993) ¹⁰	24	8.54	23	8.1	0.04
6	Pizzuli et al (1998) ¹¹	108	9.40	97	8.2	<0.001
7	Khandelwal et al (2006) ¹²	94	10.43	30	9.2	<0.001
8	Present study	39	9.30	198	7.7	<0.001

Table-3: Comparison of present study results with previous studies.

In Table 2 we combine 1a and 1b gp into gp 1 as CAD and compare with control gp. There was a significant difference in values of Hb, Platelet count, MPV, PDW (p value <0.05) (Table 2).

DISCUSSION

We compared our results with similar studies in past and shown in Table 3.⁶⁻¹²

There are potential confounding factors of MPV. It has been shown that MPV values vary between different ethnicities further more, medications and illness also influence this value for example:- Obesity, Smoking, Ageing and Diabetes increase MPV value but aspirin, clopidogrel and IBD decreased MPV.¹³⁻¹⁷ Chu et al observe stepwise decrease in MPV in subjects with chest pain in AMI, UA and non cardiac chest pain.² Yilmaz et al observed a stepwise decreased MPV between MI, UA, SCAD among patient in Turkey.¹⁸ Lipi et al reported that Italian patients with ACS had significantly higher MP values than patients without ACS.¹⁹ A few reports published have revealed a larger MPV in Indian patients with ACS compared with healthy controls or patients with SCAD.²⁰

Platelets play a pivot roll in atherogenesis.¹⁸ Platelets activation ultimately leads to the formation of thromboxan A₂, a potent vaso constrictor and platelets aggregating substance, or leucotriene, strong mediators of acute inflammatory response.¹⁹ Large hyperactive platelets play an important roll in intra-coronary thrombus formation and acute thrombotic events.²¹ Decreased in platelets count can be due to participation of platelets due to thrombus process.²² The increase in platelets consumption at the site of the coronary atherosclerotic plaque causes larger platelets to be released from the bone marrow. The fact that the increase persists even after discharge from hospital support in view that platelets volume is chronically larger in infarct group.^{7,9} This suggest that PVI, particularly MPV, are indicator of the degree of damage already done and that these marker maintain their strength and predictive value for a long time. Automated cell counter in modern hospital laboratories have made PVI routinely available. Thus, this effortless laboratory test can be add value to diagnosis of spectrum of CAD.

We found no association between the type and site of infarct and MPV, as has been reported by others.^{7,9} PC and PVI were not associated with mortality, morbidity or the severity of MI in our present study, where as martin et al found that MPV was significantly higher in those patients who died of MI, compared with survivors.⁹

The role of PDW specifically in patients with CAD and acute coronary events is yet to be explored.

Thus in our study, obtained data have confirmed the result of previous studies.⁶⁻¹² Our findings provide further evidence that platelets activation, measured by elevated MPV, may contribute to the pathogenesis of thrombosis related complications in CAD.²³

CONCLUSION

Our data suggest that the increased MPV at the admission

time is significantly higher among in patients diagnosed with AMI than in patients with SCAD or control group of same age. Because larger platelets may play a specific role in infarction and is probably a risk factor for developing coronary thrombosis and MI. patients with larger platelets can easily be identified during routine haematological analysis because PVI are generated as a byproduct of automated blood counts. Thus MPV is considered as a cost effective tool that may reflect an atherosclerotic tendency in human body.

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