ORIGINAL ARTICLE

MEAN PLATELET VOLUME (MPV) & OTHER PLATELET INDICES IN ACUTE MYOCARDIAL INFARCTION (AMI) & STABLE CORONARY ARTERY DISEASES (SCAD)

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ABSTRACT: BACKGROUND: 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. **AIM:** to study 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 clinico-pathological correlation. **SETTINGS AND DESIGN:** This was a prospective hospital based stud, carried out on 286 patients. Patients were studied in three groups, IA, IB and II. Group IA comprised of patients with AMI, IB patients with stable coronary artery disease and group II was normal healthy controls. **MATERIAL & METHODS**: This is 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. **STATISTICAL ANALYSIS USED:** Results were presented as mean, SD or frequency as appropriate. Other than these, two sample t test, one way ANOVA was used for statistical 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:** The mean platelet volume was significantly higher in patients with AMI (9.30+0.91) as compared to SCAD patients (8.38+0.85) and controls (7.71+1.05). **CONCLUSION:** Thus MPV is considered as a cost effective tool that may reflect an atherosclero-thrombotic 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 a major breakthrough affecting people worldwide including Indians. 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 a 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 plateletcrit (PCT) – routinely available in most clinical laboratories. Thus, our aim was to study 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 & METHODS: A prospective hospital based study was carried out on 286 patients over a period of 6 months. Institutional review board of the hospital approved the study protocol and informed consent was obtained from patients.

Total 286 subjects were studied in 3 groups:
Group I A: - Patients admitted to the ICU with Unstable Angina and for Acute Myocardial Ishemia.
Group I B: - Patients with Stable Coronary Artery Disease admitted for coronary angiography and coronary artery bypass grafting (CABG) procedure and follow up patients, having a previous ischemic event.
Group II: - Age and Sex matched normal healthy controls with a normal ECG.

In group IA 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 platelet parameters, hemoglobin and total leukocyte count were noticed and analyzed.

In group IA 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 IB patients were diagnosed based on the following criteria: - Evidence of AMI at least 5 weeks prior to admission. Their case reports showed:

1. Development of pathological Q wave in the ECG
2. Imaging evidence of new loss of viable myocardium or a new regional wall motion abnormality.

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 group(1B)and total 198 subjects were age and sex matched controls(group II). All hematological parameters were generated by Beckman coulter analyzer and analyzed statically. 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 Hemoglobin value in three groups. The white blood cell count was significantly raised in AMI group compared with SCAD and control group. (Table 1)

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

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

DISCUSSION: There are potential confounding factors of MPV. It has been shown that MPV values vary between different ethnicities. Furthermore, medications and illness also influence this value for example: - Obesity, Smoking, Aging and Diabetes increase MPV value but aspirin, clopidogrel and IBD decreased MPV.[6-7] 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 it MPV between MI, UA, and SCAD among patient in Turkey.[8] Lipi et al reported that Italian patients with ACS had significantly higher MP values then patients without ACS.[9] A few reports published have revealed a larger MPV in Indian patients with ACS compared with healthy controls or patients with SCAD.[10]

Platelets play a pivot roll in atherogenesis.[8] Platelets activation ultimately leads to the formation of thromboxsen A2, a potent vasoconstrictor and platelets aggregating substance, or leukotriene, strong mediators of acute inflammatory response.[9] Large hyperactive platelets play an important role in intra-coronary thrombus formation and acute thrombotic events.[11] Decrease in platelets count can be due to participation of platelets due to thrombotic process.[12] 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.[13, 14]

This suggests that PVI, particularly MPV, are indicators of the degree of damage already done and that these markers 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 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.[13, 14] PC and PVI were not associated with mortality, morbidity or the severity

of MI in our present study, whereas martin et al found that MPV was significantly higher in those patients who died of MI, compared with survivors.[14]

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

We compared our results with similar studies in past and shown in Table 3:

		AMI		CONTROLS		
	Previous studies	**N**	**MPV**	**N**	**MPV**	**P value**
1	O'Brien et al(1973)(15)	23	8.10	36	7.01	<0.001
2	Cameron et al(1983) (13)	100	9.07	200	8.32	<0.001
3	Martin et al(1983) (16)	15	7.30	22	6.32	0.05
4	Martin et al(1991) (14)	126	10.09	1590	9.72	<0.001
5	Smyth et al(1993) (17)	24	8.54	23	8.1	0.04
6	Pizzzuli et al (1998) (18)	108	9.40	97	8.2	<0.001
7	Khandelwal et al (2006) (19)	94	10.43	30	9.2	<0.001
8	Present study	39	9.30	198	7.7	<0.001

Thus in our study, obtained data have confirmed the result of previous studies.[15-19] Our findings provide further evidence that platelets activation, measured by elevated MPV, may contribute to the pathogenesis of thrombosis related complications in CAD.

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 hematological 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 atherosclerothrombotic tendency in human body.

REFERENCES:

1. Mckarns SC, Smith. CJ, Payne VM. Doolittle DJ. Blood parameters associated with atherogenic and thrombogenic risk in smokers and nonsmokers with similar life-styles. Mod Pathol 1995 May; 8(4): 434-40.
2. Chu H, Chen WL, Huang CC, Chang HY, Kuo HY, Gau CM, et al. Diagnostic performance of mean platelet volume for patients with acute coronary syndrome visiting an emergency department with acute chest pain: the Chinese scenario. Emerg Med J 2011 Jul; 28(7): 569-74.
3. Kilicli-Camur N, Demirtunc R, Konuralp C, Eskiser A, Basaran Y. Could mean platelet volume be a predictive marker for acute myocardial infarction? Med Sci Monit 2005 Aug; 11(8): 387-92.

4. Endler G, Klimesch A, Sunder-Plassmann H, Schillinger M, Exner M, Mannhalter C, et al, Mean platelet volume is an independent risk factor for myocardial infarction but not for coronary artery disease. Br J Haematol 2002 May; 117(2): 399-404.
5. Khode V, Sindur J, Kanbur D, Ruikar K, Nallukar S. Mean plateletr volume & other platelet volume indicse in patients with acute myocardial infarction, Stable coronary artery disese; a case conrol study, j cardio vasc.dis res 2012; 3: 272-5.
6. Demirtuner, Duman D, Basan M. Effects of doxazosin and amlodipine on mean platelet volume and serum serotonin level in patients with metabolic syndrome: a randomised controlled study.clin drug investig 2007; 27: 435-41
7. Kapsoitakis AN. Karkourais MI, S Firidaki A, Potamianos SP, Kosmadaki MG, Koutrouababis IE, ET AL.mean platelet volume: a useful marker infl.bowel dis activity.am j gastroententerol 2001: 96: 776-81.
8. Yilmaz MB, Cihan G, Guray Y, Guray U, Kisacik HL, Sasmaz H, et al. Role of mean platelet volume in triagging acute coronary syndromes. J Thromb Thrombolysis 2008 Aug; 26(1): 49-54.
9. Lippi G, Filippozzi L, Salvagno GL, Montagnana M, Franchini M, Guidi GC, et al. Increased mean platelet volume in patients with acute coronary syndromes. Arch Pathol Lab Med 2009 Sep; 133(9): 1441-3.
10. Smith NM, Pathansali R, Bath PM. Platelets and stroke. Vasc Med 1999; 4(3): 165-72.
11. Mathur A, Rokinson MS, Cotton J, et al. Platelet reactivity in acute coronary syndromes: evidence for differences in platelet behaviour between unstable angina and myocardial infarction. Thromb Haemost 2001; 85: 989–94.
12. Yetkin, E. Mean platelet volume not so far from being a routine diagnostic and prognostic measurement. Thromb Haemost 2008 Jul; 100(1): 3–4.
13. Cameron EA, Phillips R, Ibbatson RM, et al. Platelet size in myocardial infarction. BMJ 1983; 287: 449–51.
14. Martin JE, Bath PMW, Burr ML. Influence of platelet size on outcome after myocardial infarction. Lancet 1991; 338: 1403–11.
15. O'Brien JR, Etherington M, Jamieron S, et al. Stressed template bleeding-time and other platelet function tests in myocardial infarction. Lancet 1973; 11778: 694–6.
16. Martin JF, Plumb J, Kilbey RS, et al. Changes in volume and density of platelets in myocardial infarction. BMJ 1983; 287: 456–9.
17. Smyth DW, Martin JF, Michalis L, et al. Influence of platelet size before coronary angioplasty on subsequent restenosis. Eur J Clin Invest 1993; 23: 361–7.
18. Pizzulli L, Yang A, Martin JF, et al. Changes in platelet size and count in UA compared to stable angina or non-cardiac chest pain. Eur Heart J 1998; 19: 80–4.
19. Khandekar MM, Khurana AS, Deshmukh SD, Kakrani AL, Katdare AD, Inamdar AK. Platelet volume indices in patients with coronary artery disease and acute myocardial infarction: an Indian scenario. J Clin Pathol 2006 Feb; 59(2): 146-9.

Sr. No.		Group 1A, AMI N = 39	Group 1B, SCAD n = 49	Group 2, Controls n =198	F Test	P Value
1	Age	61.72 + 12.51	59.04 + 11.67	53.10 + 7.41	19.77	<0.0001
2	Hb	12.65 + 2.13	12.71 + 1.15	12.89 + 0.78	1.35	0.3230
3	WBC	13.24 + 4.87	8.79 + 3.57	6.15 + 1.40	128.9	<0.0001
4	Pl.Ct.	2.01 + 0.72	2.39 + 0.79	2.74 + 0.95	12.95	<0.0001
5	MPV	9.30 + 0.91	8.38 + 0.85	7.71 + 1.05	44.23	<0.0001
6	PDW	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.		Group 1 CAD n = 88	Group 2, Controls n =198	T Test	P Value
1	Age	60.23 + 12.05	53.10 + 7.41	6.126	<0.0001
2	Hb	12.68 + 1.65	12.89 + 0.78	- 1.489	0.138
3	WBC	10.76 + 4.72	6.15 + 1.40	12.592	<0.0001
4	Pl.Ct.	2.22 + 0.78	2.74 + 0.95	- 4.639	<0.0001
5	MPV	8.70 + 0.99	7.71 + 1.05	8.128	<0.0001
6	PDW	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

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