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Correlation between MPV and Lipid Profile in Eastern India: A Cross Sectional Study

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ABSTRACT

MPV measures the average size of platelets and is an emerging risk factor of atherosclerosis. The functions of platelets include adhesion, shape change and spreading, aggregation, secretion, procoagulant activity and clot retraction. Mean Platelet Volume can be simply analyzed by the ABX pentra automated analyzer using electrical impedance. There are many markers of atherosclerosis. The present study aims to find a correlation of MPV with Total Cholesterol, LDL, HDL,VLDL,Triglyceride.

Aims and Objectives: To determine the correlation between MPV and Lipid Profile (Total cholesterol,LDL,HDL,VLDL,Triglyceride)

Materials and methods: 150 patients of Department of General Medicine participated in the study. The patients were selected by simple random sampling. Inclusion and exclusion factors were considered. Patients above 18 years of age were selected. Patients taking medications that reduce platelets, alcoholics and those having hereditary disorders of large platelets were excluded.

Results: MPV is inversely correlated with HDL . Thus methods applied to increase HDL in general population will lead to decrease in MPV and lower risk of atherosclerosis. There is a positive correlation of MPV with VLDL and Triglyceride, but the correlations are not statistically significant. There is a negative correlation of MPV with LDL and Total Cholesterol, but the correlations are not statistically significant.

Key Words: MPV, Lipid profile, HDL

INTRODUCTION

Platelet size, measured as mean platelet volume (MPV), is a marker of platelet function and is positively associated with indicators of platelet activity, including aggregation and release of thromboxane A2, platelet factor 4, and β-thromboglobulin[1,2]. In normal individuals the platelet count is inversely proportional to MPV; platelet mass (the product of MPV and platelet count) is a near constant.

Platelets, or thrombocytes (from Greek θρόμβος, "clot" and κύτος, "cell"), are small, irregularly shaped clear cell fragments (i.e. cells that do not have a nucleus), 2–3 μm in diameter,[3] which are derived from fragmentation of precursor megakaryocytes.

Megakaryocytes arise from pluripotent stem cell that develops into 2 types of precursors, burst-forming cells and colony-forming cells, both of which express the CD34 antigen [4]. Thrombopoietin (TPO), the primary regulator of thrombopoiesis, is currently the only known cytokine required for megakaryocytes to maintain a constant platelet mass (though TPO is not increased on platelet destruction). TPO is thought to act in conjunction with other factors, including IL-3, IL-6, and IL-11, although these cytokines are not essential for megakaryocyte maturation [4].

When aging, platelets contain decreased levels of sialic acid and they accumulate surface IgG which function in removal of old platelets [5]. Senescent platelets are removed primarily by macrophages in the spleen, although the larger blood flow

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through the liver allows severely damaged platelets to be removed more quickly by hepatic macrophages [6]

Although platelets are incapable of de novo protein synthesis they are very active metabolically and respond rapidly to vascular injury or trauma by undergoing a series of reactions (adhesion, release of granule contents, shape change and aggregation), which ultimately result in the formation of a platelet–fibrin plug.

Platelets bud off megakaryocytes in the marrow. Platelet size and volume (e.g. Mean Platelet Volume) depends on the circumstances of their production in the marrow. MPV is **not** related to aging of platelets in the circulation. Platelet parameters are very stable in most patients. MPV is increased in conditions with *increased* platelet production eg. immune thrombocytopenia, disseminated intravascular coagulation, myeloproliferative disorders, pre-eclampsia and recovery from transient hypoplasia (cytotoxic chemotherapy). MPV is decreased in conditions associated with *under* production of platelets e.g. Bone marrow aplasia.

Mean Platelet Volume (MPV) correlates with the functional status of platelets and is an emerging risk marker for atherothrombosis [7]. There is evidence that platelet function is accentuated in acute ischemic stroke [8]. Increased mean platelet volume (MPV), indicating higher platelet reactivity, is associated with an increased risk of myocardial infraction.

MPV appears to be a marker, or even a determinant, of platelet function. Large platelets are more reactive than small platelets in vitro. Large platelets differ from normal or small platelets in the following ways [9];

i. They preferentially and more rapidly aggregate to platelet agonist including ADP, collagen and adrenaline.
ii. They produce more prothrombotic and vasoactive factors including arachidonic acid metabolites (e.g. Thromboxane A2), serotonin, β thromboglobulin and ATP.
iii. They contain more dense granules.
iv. They have higher LDH activity.
v. They are associated with a decreased bleeding time (BT; a measure of in vivo haemostatic function) [8].
vi. MPV correlates with platelet aggregation, whether measured in platelet rich plasma or whole blood.
vii. Large platelets also express increased levels of adhesion molecules. eg. P- selectin, GPIIb/IIIa although the surface density of these glycoproteins is usually constant independent of platelet volume.[10]

MATERIAL AND METHODS

STUDY SETTING
Indoors and Out Patient Department of General Medicine, R. G. Kar Medical College and Hospital, Kolkata which is a tertiary care referral centre

DURATION OF STUDY
One year

STUDY POPULATION
150 patients of Department of General Medicine after taking Ethical clearance and signing consent form .

INCLUSION CRITERIA
1. Gender: Males/Females
2. Age Range: 18 years and above
3. Socioeconomic group: All

EXCLUSION CRITERIA
1. Known cases of hereditary disorders of large platelets.
2. Medications that reduce platelets
3. Alcoholic

SAMPLING
SIMPLE RANDOM SAMPLING

CONTROLS REQUIRED
No

STUDY DESIGN
Descriptive Cross Sectional study

PARAMETERS TO BE STUDIED
1. Sociodemographic parameters: Mean Age, percentage of patients, male/female, of urban/rural area, different religion, and different occupation, sedentary or active life style and type of diet intake.
2. Clinical parameters
   Blood pressure, BMI, Waist Hip Ratio
3. Hematological parameters: Mean Hemoglobin (Hb), Total leukocyte count (TLC), Differential leukocyte count (DLC), Platelet Count, and Mean Platelet Volume (MPV).
4. Biochemical parameters: Mean serum urea, creatinine, Bilirubin, Total protein, Albumin, SGOT, SGPT, Glucose (fasting and post prandial), Serum electrolytes, Complete lipid profile

Method of MPV Measurement
A Blood sample was collected from the antecubital vein using a 5cc syringe and transferred to an EDTA vacutainers. The samples were then taken to the laboratory after storage at room temperature for 2 hours but before 4 hours of collection and analyzed using the ABX pentra automated analyzer using electrical impedance to measure the mean platelet volume. After the analysis the same sample was taken to the central laboratory and a peripheral smear was done to look for platelet aggregates. If platelet aggregates were found then such cases were excluded from the study.

The normal range of Mean Platelet Volume measured in EDTA blood is 7.8-11 fl. Values of 11.1 fl and above are considered as abnormally high [11].

DATA ANALYSIS

The Statistical software namely SPSS 20.0, Stata 8.0, MedCalc 9.0.1 and Systat 11.0 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc.

STATISTICAL METHODS

Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented on Mean ± SD (Min-Max) and results on categorical measurements are presented in Number (%). Significance is assessed at 5 % level of significance. Student t test (two tailed, independent) has been used to find the significance of study parameters on continuous scale between two groups.

Limitation

Small sample size

RESULTS AND ANALYSIS

The maximum number of patients belonged to age group 56 to 65 years (38%) ,the second highest number belonged to age group 46 to 55 years (32%) and minimum number were in the under 25 group

In the study population 56% are male and 44% are female.

Table 1: Correlation of MPV with Haemoglobin

		Mean Platelet Volume	Haemoglobin
Mean Platelet Volume	Pearson Correlation	1	-.054
	Sig. (2-tailed)		.514
	N	150	150
Haemoglobin	Pearson Correlation	-.054	1
	Sig. (2-tailed)	.514	
	N	150	150

Comments: There is a negative correlation of MPV with Hemoglobin and the correlation is not statistically significant.

Table 2: Correlation of MPV with Total Leucocyte Count

		Mean Platelet Volume	Total Leucocyte Count
Mean Platelet Volume	Pearson Correlation	1	-.026
	Sig. (2-tailed)		.753
	N	150	150
Total Leucocyte Count	Pearson Correlation	-.026	1
	Sig. (2-tailed)	.753	
	N	150	150

COMMENTS: There is a negative correlation of MPV with Total Leucocyte Count and the correlation is not statistically significant.

Table 3. Correlation of MPV with HDL

		Mean Platelet Volume	HDL
Mean Platelet Volume	Pearson Correlation	1	-.181*
	Sig. (2-tailed)		.026
	N	150	150
HDL	Pearson Correlation	-.181*	1
	Sig. (2-tailed)	.026	
	N	150	150

*. Correlation is significant at the 0.05 level (2-tailed).

COMMENT: There is a negative correlation of MPV with HDL and the correlation is statistically significant.

Table 4: Correlation of MPV with VLDL

		Mean Platelet Volume	VLDL
Mean Platelet Volume	Pearson Correlation	1	.099
	Sig. (2-tailed)		.226
	N	150	150
VLDL	Pearson Correlation	.099	1
	Sig. (2-tailed)	.226	
	N	150	150

COMMENTS: There is a positive correlation of MPV with VLDL and the correlation is not statistically significant

Table 5: Correlation of MPV with LDL

		Mean Platelet Volume	LDL
Mean Platelet Volume	Pearson Correlation	1	-.013
	Sig. (2-tailed)		.876
	N	150	150
LDL	Pearson Correlation	-.013	1
	Sig. (2-tailed)	.876	
	N	150	150

COMMENT: There is a negative correlation of MPV with LDL and the correlation is not statistically significant.

Table 6: Correlation of MPV with Triglycerides

		Mean Platelet Volume	TRIGLYCERIDES
Mean Platelet Volume	Pearson Correlation	1	.100
	Sig. (2-tailed)		.223
	N	150	150
Triglycerides	Pearson Correlation	.100	1
	Sig. (2-tailed)	.223	
	N	150	150

COMMENT: There is a positive correlation of MPV with Triglycerides and the correlation is not statistically significant.

Table 7: Correlation of MPV with Total Cholesterol

		Mean Platelet Volume	Total Cholesterol
Mean Platelet Volume	Pearson Correlation	1	-.003
	Sig. (2-tailed)		.973
	N	150	150
Total Cholesterol	Pearson Correlation	-.003	1
	Sig. (2-tailed)	.973	
	N	150	150

COMMENT: There is a negative correlation of MPV with Total Cholesterol and the correlation is not statistically significant.

Table 8: Correlation of MPV with Platelet Count

		Mean Platelet Volume	Platelet Count
Mean Platelet Volume	Pearson Correlation	1	-.171*
	Sig. (2-tailed)		.036
	N	150	150
Platelet Count	Pearson Correlation	-.171*	1
	Sig. (2-tailed)	.036	
	N	150	150

*. Correlation is significant at the 0.05 level (2-tailed).

COMMENT: There is a negative correlation of MPV with Platelet Count and the Correlation is statistically significant.

DISCUSSION

When correlating MPV with clinical and laboratory parameters, MPV had statistically insignificant correlations with Hemoglobin (p=0.514), Total Leukocyte Count (p=0.753), VLDL (p=0.226), LDL (p=0.876), Triglyceride(p=0.223) and Total Cholesterol (p=0.973).

MPV had a negative correlation with HDL (Pearson coefficient = -0.181) and the correlation is significant at the 0.05 level (2-tailed)

(p value=0.026).

MPV had a negative correlation with Platelet Count (Pearson coefficient = -0.171) and the correlation is significant at the 0.05 level (2-tailed) (p value=0.036). This finding correlates with findings of previous studies where it is shown that as platelet mass remains constant in a given person, the platelet count decreases as the MPV increases (Platelet Mass = MPV × Platelet Count).

CONCLUSION

Platelets contribute to atherosclerotic complications and lead to thrombus formation [12].

This study has shown that MPV has inverse correlation with HDL. Thus, interventions targeted towards increasing HDL in population will lead to decreased MPV and thus decreasing the incidence of atherosclerotic diseases.

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CONTRIBUTION OF AUTHORS

1. Mukherjee Biswaroop - Concept and Study Design, writing the paper
2. Mallik Sreya - Data Collection and writing the paper
3. Maitra Somnath - Statistical Analysis, writing the paper and being the Corresponding Author

REFERENCES

1. Sharp DB, Bath PMW, Martin JF, et al. Cigarette smoking sensitizes and desensitizes impedance-measured ADP-induced platelet aggregation in whole blood. Thromb Haemost. 1995;74:730–735.
2. D'Erasmo E, Aliberti G, Celi F, et al. Platelet count, mean platelet volume and their relation to prognosis in cerebral infarction. J Intern Med.1990;227:11–14.
3. Campbell, Neil A.(2008).Biology(8th ed.). London: Pearson Education. p. 912
4. Patel SR, Hartwig JH, Italiano JE Jr. The biogenesis of platelets from Megakaryocyte proplatelets. The Journal of Clinical Investigation 2005; 115: 3348–3354.
5. George JN. Platelet IgG: measurement, interpretation, and clinical significance. ProgHemost Thromb.1991; 10:97-126.
6. Buckley MF, James JW, Brown DE, Whyte GS, Dean MG, Chesterman CN, et al. A novel approach to the assessment of variations in the human platelet count. Thromb Haemost 2000; 83: 480-484.
7. *Arch Pathol Lab Med*. Vol 133, September 2009;1441-43
8. Smith N, Pathansali R, Bath P. Platelets and stroke. Vascular medicine 1999; 4:165-172.
9. Bath PMW, Butterworth RJ. Platelet size: measurement, physiology and vascular disease. Blood1996; 7: 157–61.
10. Victor M, Ropper AH, Adams RD. Cerebrovascular diseases. Adam's and Victor's Principles of Neurology. Ed. Ropper A H, Samuels MA. 9th ed. United States of America, McGraw Hill 2009. Smith N, Pathansali R, Bath P. Platelets and stroke. Vascular medicine 1999; 4:165-172.
11. *Jam Coll Cardiol*. 2013;62(18_S2):C116-C116. doi:10.1016/j.jacc.2013.08.354
12. Greisenegger S, Endler G, Hsieh K, Tentschert S, Mannhalter C, Lalous chek W, Is elevated mean platelet volume associated with a worse outcome in patients with acute ischemic cerebrovascular events? Stroke 2004;35:1688–91.