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A Study of platelet volume indices in patients of coronary artery diseases

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Abstract

Objectives: 1. To investigate platelet indices in patients of Myocardial infarction, Unstable angina, and Stable angina. 2. To compare changes in platelet indices among Patients of myocardial infarction, unstable angina, stable angina and normal population. **Material and Methods:** This study was carried out in the Department of Medicine, NIMS Medical College and Hospital, Jaipur, Rajasthan. To investigate the values of platelet indices MPV, PDW and platelet count with the view of exploring their use in predicting the possibility of impending events of coronary artery disease on 110 patients admitted in wards and ICU under Department of Medicine and 110 controls who were the normal during the study period. **Results:** On comparing patients of coronary artery disease with control group it was found that MPV and PDW were raised and platelet counts decreased with a highly significant difference ($p<0.001$). **Conclusion:** Platelet morphology played an important role in thrombosis and this fact could be used therapeutically in future.

Keywords: Mean platelet volume, Platelet distribution width, Platelet indices, Coronary artery disease..

INTRODUCTION

Vascular diseases are leading cause of morbidity and mortality in India and worldwide of which coronary artery disease is common.[1] Atherosclerosis is a systemic inflammatory process characterised by the accumulation of lipids and macrophages/lymphocytes within the intima of large arteries. These initial fatty streak lesions may also evolve into vulnerable plaques susceptible to rupture or erosion. Plaque disruption initiates both platelet adhesion and aggregation on the exposed vascular surface and the activation of the clotting cascade leading to the so-called atherothrombotic process.[2]

Platelets not only act as mediators of thrombus formation, but also they are potent inflammatory cells that induce inflammatory responses in adjacent cells such as leukocytes and endothelial cells. They may also themselves respond to inflammatory mediators produced by these neighboring cells. The bidirectional interaction between platelets and other cells may also be involved in the non resolving inflammation characterizing atherosclerosis.[3]

Altered platelet morphology and function are likely to be associated with increased risk of vascular disease. Large platelets contain more dense granules, are metabolically and enzymatically more active than small platelets and produce more thromboxane A2[4]. They release more serotonin and β-thromboglobulin, hence having higher thrombotic potential[5.] MPV (Mean platelet volume) showing platelet size reflects platelet function and activity. Its value is around 7.4-10.4fL[6]. Platelets which are large in size i.e. ones which have higher MPV contribute to thrombosis to a greater extent as compared to small platelets hence is a marker of hypercoagulability[7]. MPV is higher when there is destruction of platelets[.8] PDW (Platelet distribution width) provides information about the range of platelet size in a blood sample. It increases during platelet activation as in coronary artery disease where more number of large platelets are found. It is a more specific marker of platelet activation than MPV since it does not increase during simple platelet swelling[9].

In this study the values of platelet indices – MPV, PDW and platelet count were observed in patients of coronary artery disease with a view to explore their use in predicting the possibility of impending events.

MATERIAL AND METHODS

This study was carried out in the Department of Medicine, NIMS Medical College and Hospital, Jaipur, Rajasthan. The study was conducted on 110 patients admitted in wards and ICU under Department of Medicine and 110 controls who were the normal and healthy during the study period. These subjects were divided into groups A and B.

Group A- patients with Coronary Artery Disease

Myocardial Infarction-50 patients, Unstable Angina-40 patients, Stable Angina – 20 patients

Group B – 110 age and sex matched normal healthy controls.

Patients with primary platelet disease, any bleeding disorder, any clotting disorder,Patients taking antiplatelet therapy, Patients on statins and doxazosin were not included. Blood samples were obtained before giving any antiplatelet or anticoagulant therapy. Blood was collected in dipotassium EDTA tubes by a clean puncture avoiding bubbles and froth. Sample was examined by running into autoanalyser (Benesphera three part Hematology Analyser H31) within two hours of venepuncture.

Normal value of platelet count is 1,50,000 to 4,50,000/μL[10]; PDW is 9 to 14 fL and MPV is 7.4 to 10.4 fL.[6]

Complete history regarding name, age, sex, occupation, socioeconomic status, hypertension, diabetes, smoking, antiplatelet therapy, bleeding disorder, clotting disorder, platelet disease was taken and complete physical and general examination was done. Various investigations like complete hemogram, Peripheral blood film, Platelet volume indices- Mean platelet volume, platelet distribution width; Urine examination complete, Blood Urea, Serum Creatinine, Random blood sugar, Serum electrolyte, Lipid profile, CPK-MB, Troponin T, ECG, Chest X ray and 2d-Echocardiography was done.

Statistical Analysis

Statistical tests like Chi- square test, unpaired 't' test and ANOVA test were applied . A p value <0.05 was considered statistically significant. Data obtained was analysed statistically by SPSS software.

OBSERVATIONS AND RESULTS

Table 1: Comparison of groups with respect to Platelet Indices

Platelet Indices	Group	N	Mean	Std. Deviation	'p' Value*
MPV(in fL)	CAD	110	10.05	1.01	<0.001
	Control	110	8.14	0.72	
PDW (in fL)	CAD	110	14.68	1.02	<0.001
	Control	110	10.71	0.48	
Platelet Count ($\times 10^9$/L)	CAD	110	264	33	<0.001
	Control	110	285	50	

*Unpaired 't' test

On comparing patients of coronary artery disease with control group it was found that MPV and PDW were raised and and platelet counts decreased with a highly significant difference (p<0.001).

Table 2: Comparison of groups with respect to MPV

Group	N	Mean	Std. Deviation	'p' Value*
Unstable Angina	40	10.19	0.77	0.632
MI	50	10.29	1.12	
Unstable Angina	40	10.19	0.77	<0.001
Stable Angina	20	9.19	0.62	
MI	50	10.29	1.12	<0.001
Stable Angina	20	9.19	0.62	

*Unpaired 't' test

MPV values were raised with a highly significant difference (p<0.001) when patients of myocardial infarction group and unstable angina group were compared with patients of stable angina. However, difference was not significant (p>0.05) on comparing patients of myocardial infarction with unstable angina.

Table 3: Comparison of groups with respect to PDW

Group	N	Mean	Std. Deviation	'p' Value*
Unstable Angina	40	14.85	0.70	0.132
MI	50	15.11	0.88	
Unstable Angina	40	14.85	0.70	<0.001
Stable Angina	20	13.25	0.44	
MI	50	15.11	0.88	<0.001
Stable Angina	20	13.25	0.44	

*Unpaired 't' test

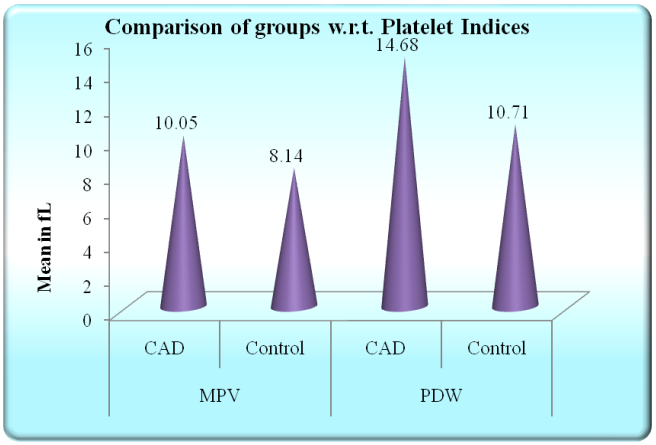
The values of PDW were raised with a highly significant difference (p<0.001) when patients of myocardial infarction group and unstable angina group were compared with patients of stable angina. However, difference was not significant (p>0.05) on comparing patients of myocardial infarction with unstable angina.

Table 4: Comparison of groups with respect to Platelet Count

Group	N	Mean	Std. Deviation	'p' Value*
Unstable Angina	40	261	31	0.884
MI	50	260	33	
Unstable Angina	40	261	31	0.049
Stable Angina	20	279	36	
MI	50	260	33	0.038
Stable Angina	20	279	36	

*Unpaired 't' test

Platelet counts were lower in myocardial infarction and unstable angina groups as compared to stable angina group .The difference was just significant on comparing myocardial infarction with stable angina(p=0.038) and unstable angina with stable angina patients (p=0.049). The difference was not significant (p>0.05) on comparing patients of myocardial infarction and unstable angina.



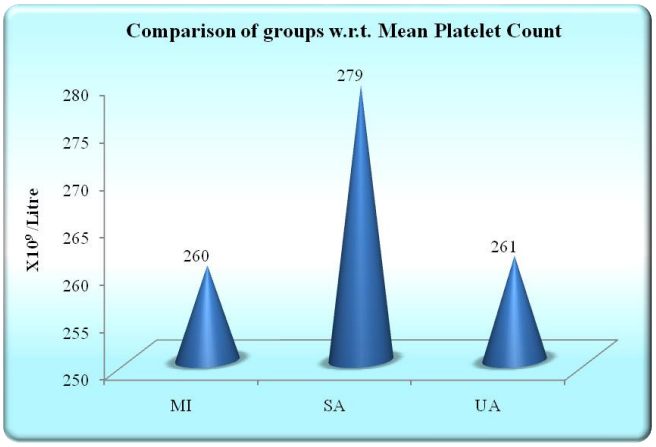


Figure 1: Comparisons of groups w.r.t mean platelet indices and mean platelet count

DISCUSSION

Platelets are heterogeneous blood elements with diverse sizes and densities. They modulate important pathophysiological processes including inflammation and coagulation. Platelets play a crucial role in pathogenesis of atherosclerotic complications contributing to thrombus formation or apposition after plaque rupture.[11] Larger and hyperactive platelets play a pivotal role in accelerating the formation and propogation of thrombus, leading to the occurrence of acute thrombotic events.[12]

The increase in platelet consumption at the site of the atherosclerotic plaque causes larger platelets to be released from the bone marrow.[13] This leads to larger mean platelet volume and lower platelet count in coronary and cerebral ischemia. But there are studies indicating that platelet size is determined at the level of the progenitor cell. Megakaryocyte ploidy is influenced by interleukin 3 and interleukin 6, which leads to the production of larger platelets that are more reactive.[14,15]

In this study we found that MPV and PDW is significantly raised in cases compared to controls.

Our study was further supported by studies conducted by Khandekar et al in 2006[13], Lievens and von Hundelshausen in 2011[16], Gupta *et al* in 2012[17].

Table 5: Comparison of MPV and PDW in CAD, MI, unstable angina, stable angina

Disease	Platelet distribution width in fl (PDW)		Mean platelet volume in fl (MPV)	
	Cases	Controls	Cases	controls
Coronary artery disease (p<0.001)	14.68±1.02	10.71±0.48	10.05±1.01	8.14± 0.72
Myocardial infarction (p<0.001)	15.11±0.88	10.71±0.48	10.29 ± 1.12	8.14± 0.72
Unstable angina(p<0.001)	14.85±0.70	10.71±0.48	10.19±0.77	8.14± 0.72
Stable angina(p<0.001)	13.25 ± 0.44	10.71±0.48	9.19± 0.62fL	8.14± 0.72

CONCLUSION

In this study it was found that large platelets contribute to the prethrombotic state in ischemic syndromes and they may play a specific role in infarction. Patients with larger platelets can easily be identified during routine haematological analysis. Hence, these indices serve as an important and cost effective tool in predicting an impending ischemic event.

It was observed in this study that platelet morphology played an important role in thrombosis and this fact could be used therapeutically in future. Presently therapeutic implications regarding platelets are restricted to their numbers only. Whereas, it may be important to modify their morphology. Trials are going on to study the effect of various drugs on platelet morphology so that we can have a new line of preventive treatment for thrombotic events.

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