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Investigation of the Association Between Mean Platelet Volume and Hemoglobin A1Cin Patients with Type II Diabetes

Mallem Babu[1], N.N. Anand[2]

[1]Assistant Professor, Department of General Medicine, SPMCW, SVIMS, Tirupati, Andhra Pradesh, India.
[2]Professor, General Medicine, Balaji Medical College, Chennai, India.

Abstract
Background: To investigate MPV in individuals with type 2 diabetes and to determine whether there is a correlation between MPV and the level of diabetic control measured by HbA1c.
Material and Methods: From December 2015 to September 2016 the case control research was carried out at General Medicine, Balaji Medical College, Chennai, India. As subjects, 60 Type 2 Diabetes Mellitus sufferers visiting Medicine OPD including those hospitalised to the hospital with at least six months of duration of illness were selected. A sample of 40 healthy controls with similar ages and sexes was taken.
Results:FBS levels were 1.31 times higher in uncontrolled diabetes vs. controlled diabetes, 1.32 times higher in diabetes vs. non-diabetics, and 1.73 times higher in uncontrolled diabetes vs. non-diabetics. Uncontrolled diabetes patients had 1.29 times higher PPBS levels than those with controlled diabetes, 1.52 times higher than non-diabetics, and 1.96 times higher than non-diabetics. Uncontrolled diabetes had a 1.12-fold greater platelet count than controlled diabetes, 1.58-fold higher than non-diabetics, and 1.68-fold higher than non-diabetics. MPV levels correlated at 5% with FBS, PPBs, and HbA1c.
Conclusion:FBS levels of treated and uncontrolled diabetics were greater than non-diabetics. Patients with managed and uncontrolled diabetes had greater PPBS levels than non-diabetics. Managed and uncontrolled diabetes patients had greater platelet counts than non-diabetics. Patients with treated and uncontrolled diabetes had larger mean platelet volume than non-diabetics. MPV levels correlated at 5% with FBS, PPBs, and HbA1c.
Keywords: Mean platelet volume, HbA1c, diabetes.

Corresponding Author: Dr.MallemBabu, Assistant Professor, Department of General Medicine, SPMCW, SVIMS, Tirupati, Andhra Pradesh, India.

Introduction

To understand vascular disease in diabetes, you must understand platelets' role in haemostasis. MPV possesses hemostatic properties. Large platelets can perform haemostatic, vasomotor, and pro-inflammatory tasks more efficiently than tiny platelets because they are more reactive, have more granules, release more serotonin and -thromboglobulin, and aggregate more easily. Higher MPV is linked to ADP and collagen-induced in vitro aggregation.[1-3] Increased platelet volume is linked to acute cerebral ischemia, TIA, AMI, and chronic vascular disease. Myocardial infarction and stroke survivors do worse with a high MPV. Platelets aid in the production of thrombi or apposition after plaque rupture, which contributes to atherosclerosis. Smokers and hypercholesterolemic individuals have larger platelets, according to research. Mean Platelet Volume (MPV), Platelet Distribution Width (PDW), and Platelet Large Cell Ratio help us understand platelet function and morphology (P- LCR).[4-6]

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

MPV and PDW are easy-to-measure platelet indicators that rise after activation. Platelets expand when activated. Pseudopodies grow and change shape into spheres. PDW is 8-12 fL compared to MPV. The "P-LCR" ranges from 15 to 35% for large platelets with a volume of 12 fL. Reduced insulin production and tissue insulin resistance characterise type 2 diabetes. Diabetes patients have multifactorial platelet hyperactivity and higher baseline activation due to hyperglycemia, hyperlipidemia, insulin resistance, and inflammatory and oxidative state. Increased platelet activity caused by inappropriate insulin action reveals vascular problems. Diabetic patients have higher MPV levels and micro vascular problems such retinopathy and micro albuminuria.[6-8]

Hyperglycemia increases platelet reactivity both directly and via glycating platelet proteins. Platelets from type 2 diabetics agglomerate faster and adhere to vascular endothelium. Platelet activity is limited by prostacyclin and nitric oxide produced by vascular endothelium. Diabetes causes increased thromboxane and/or prostacyclin production and hyperreactivity. Insulin reduces platelet activity. Platelet sensitization increases PGI2 and NO production. Diabetes causes disorganised platelets due to lack of insulin action, which promotes macrovascular and microvascular events.[8-10]Papanas, Hekimsoy, and Zuberi discovered an increase in MPV. Diabetes increases MPV in all studies. Ate O et al. found comparable outcomes in diabetic patients. We coupled diabetes control (as measured by HbA1c) with MPV because few studies in this region of the country employ both.

Material and Methods

The research took place from December 2015 to September 2016 at General Medicine, Balaji Medical College, Chennai, India. 60 Type 2 Diabetes Mellitus patients with at least 6 months of illness were studied. 40 healthy age- and-sex-matched controls were used. All subjects gave written, informed consent. Before conducting the study, ethical approval was obtained.

Inclusion criteria

1. Either gender,
2. Over 30 yr age
3. Type 2 Diabetes Mellitus patients diagnosed using ADA 2013 criteria:

Exclusion criteria: Subjects with these conditions

1. Antiplatelet drug users
2. ITP
3. Septicaemia
4. Pregnancy
5. Other life-threatening conditions
6. ESRD
7. Cirrhosis
8. Fulminant hepatic failure
9. Men with Hb less than 11gm% and women with Hb less than 10gm% because nutritional anaemia might produce reactive thrombocytosis and elevated MPV.

Methodology

Sample Analysis on admission, blood was drawn into dipotassium EDTA tubes to prevent clotting and bubble formation. The sample will be passed through a Sysmex XT-2000 automatic cell counter two hours after venipuncture. The glucose oxidase in an autoanalyzer (Hitachi 902) was used to calculate FBS and PPBS, while ion exchange chromatography was used to calculate HbA1c.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

Patients' complete medical histories were taken, including but not limited to: age, sex, smoking, alcohol use, diabetes duration, treatment, family history of diabetes, and numbness, weakness in limbs suggestive of peripheral neuropathy. Patients' vitals were taken, including their height, weight, body mass index, and waist-to-hip ratio, and they were all given a thorough physical and systemic examination.

Participants were subjected to a series of tests in a laboratory. Hemoglobin, complete blood count, differential, platelet, and erythrocyte sedimentation rates, Measures of Platelet Volume (MPV and PDW) (PDW), Urea and Creatinine. Combine PPBS with a fasting regimen, HbA1c. HDL, LDL, VLDL lipids and triglycerides [10,11].

Statistical tests were used for both descriptive analysis and comparison of the data. analysis using unpaired and paired t tests performed, while categorical data analysis by using Chi-Square and Fisher Exact tests. Correlation determined using both analysis of variance and pearson's correlation. P< 0.05 was the threshold for significance. EpiInfo (version 7.1.0.6; CDC, USA) and Excel 2010 were used to examine the data.

RESULTS

Table 1: HbA1c Distribution - Groups

Groups	**A group**	**B group**	**Control**
Description	Diabetics with HBA1c ≤7%	Diabeticswith HBA1c > 7%	Non-Diabetics
Number	24	36	40
Mean	6.48	10.03	5.82
SD	0.34	2.96	0.74
Group		**Intervention**	
Group A		Diabetes under control	
Group B		Uncontrolled Diabetes	
Control Group		Non-Diabetic	

Table 2: Age - Groups

Age -Groups	**Group A**	**Group B**	**Control**	**Group A (%)**	**Group B (%)**	**Control (%)**
31-40years	5	7	2	20.83	19.44	5.00
41-50years	5	11	17	20.83	30.56	42.50
51-60years	10	10	18	41.67	27.78	45.00
61-70years	4	8	3	16.67	22.22	7.50
Total	24	36	40	100	100	100

Table 3: Distribution of Age

Distribution of Age	**Group A**	**Group B**	**Control**
Mean	50.92	50.50	50.85
SD	9.91	11.18	6.06
P Value andUnpaired t Test	Group A Vs Group B		0.8830
	Group A Vs Control		0.9734
	Group B Vs Control		0.8639

Patients in Group A had a mean age of 50.92 (n=10, 41.67%). The mean age of the participants in Group B was 50, and the majority were between the ages of 41 and 50 (30.56%). The average age of patients in the Control Group was 50.85. There were 18 of

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

them, or 45% of the total. No statistically significant difference in age in the study groups by an unpaired t test ($p > 0.05$).

All 12 patients in Group A were of even sexes. There were more women (55.56%) than men (44.44%) in Group B. There were more women than men in the Control Group (n=22 vs. n=18, or 55% female to 45% male). Chi-square analysis shows no significant correlation between the gender in the research groups. [Table 4]

Table 4: Gender Status

Gender Status	GroupA	GroupB	Control	GroupA (%)	GroupB (%)	Control(%)
Male	12	16	18	50.00	44.44	45.00
Female	12	20	22	50.00	55.56	55.00
Total	24	36	40	100	100	100
P Value Chi Squared Test				0.9021		

Table 5: FBS – Groups

FBS -Groups	Group A	Group B	Control	Group A (%)	Group B (%)	Control (%)
≤ 100mg/dL	8	1	35	33.33	2.78	87.50
101 -150mg/dL	13	17	5	54.17	47.22	12.50
151-200mg/dL	3	15	0	12.50	41.67	0.00
> 200mg/dL	0	3	0	0.00	8.33	0.00
Total	24	36	40	100	100	100

Table 6: Distribution of FBS

FBS Distribution	Group A	Group B	Control
Mean	118.38	154.75	89.48
SD	24.08	34.99	9.70
P Value Unpaired t Test	Group A Vs Group B		<0.0001
	Group A Vs Control		<0.0001
	Group B Vs Control		<0.0001

Mean FBS levels in group A were 118.38 mg/dL (range: 101-150 mg/dL), with 13 out of 18 patients (54.17%) falling into this range. The median FBS level in group B was 154.75 mg/dL, with the vast majority of patients (n=17, 47.22%) falling in the range of 101–150 mg/dL. FBS levels in the control group averaged 89.48 mg/dL (n=35, 87.50%).

Table 7: Group- PPBS

PPBS - Groups	GroupA	GroupB	Control	GroupA (%)	GroupB (%)	Control(%)
≤ 150 mg/dl	14	5	40	58.33	13.89	100.00
151-200 mg/dl	4	14	0	16.67	38.89	0.00
201-250 mg/dl	4	9	0	16.67	25.00	0.00
251-300 mg/dl	2	4	0	8.33	11.11	0.00
> 300 mg/dl	0	4	0	0.00	11.11	0.00
Total	24	36	40	100	100	100

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

Table 8: Distribution of PPBS

Distribution of PPBS	Group A	Group B	Control
Mean	165.71	213.39	109.00
SD	49.24	59.42	10.79
P Value Unpaired t Test	Group A Vs Group B		0.0019
	Group A Vs Control		<0.0001
	Group B Vs Control		<0.0001

The average PPBS level for patients in group A was 165.71 mg/dl (n=14, 58.33%). Patients in group B (n=14, 38.89%) had a mean PPBS level of 213.39 mg/dl, with the majority having a level between 151 and 200. The majority of patients (n=40, 100%) in the control group had PPBS levels greater than 150 mg/dl (mean: 109 mg/dl).

Table 9: Groups- Platelet count

Platelet Count - Groups	Group A	Group B	Control	Group A (%)	Group B (%)	Control (%)
≤ 250(X 10 /L)	8	4	27	33.33	11.11	67.50
251-300(X 10 /L)	12	21	13	50.00	58.33	32.50
301-350(X 10 /L)	4	11	0	16.67	30.56	0.00
Total	24	36	40	100	100	100

Table 10: Distribution of Platelet count

Distribution of Platelet count	Group A	Group B	Control
Mean	267.20	284.00	243.03
SD	23.72	27.87	21.11
P ValueUnpaired t Test	Group A Vs Group B		0.0185
	Group A Vs Control		0.0001
	Group B Vs Control		<0.0001

With a mean platelet count of 267.20 X 10 /L, and a range of 251 to 300 X 10 /L (n=12, 50%), the majority of patients in group A fell within the middle range. Among group B, the median platelet count was 284 (range: 251-300 X 10 /L), and the majority of patients (n=21, 58.33%) had a platelet count between 251 and 300. The majority of patients in the control group (n=27, 67.50%) had platelet counts of 250 X 10 /L or higher. The correlation between study groups and platelet count is statistically significant (p <0.05), as determined by an unpaired t test comparing groups A and B, as well as groups A and the control group.

Table 11: Groups - MPV

Groups –MPV	Group A	Group B	Control	Group A (%)	Group B (%)	Control (%)
≤ 8.00 fL	3	0	18	12.50	0.00	45.00
8.01-10.00 fL	15	9	22	62.50	25.00	55.00
10.01-12.00 fL	6	24	0	25.00	66.67	0.00
> 12 fL	0	3	0	0.00	8.33	0.00
Total	24	36	40	100	100	100

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

Table 12: Distribution of MPV

Distribution of MPV	Group A	Group B	Control
Mean	9.55	10.65	7.98
SD	0.99	0.88	0.41
P ValueUnpaired t Test	Group A Vs Group B		<0.0001
	Group A Vs Control		<0.0001
	Group B Vs Control		<0.0001

The average MPV for group A patients was 9.55 fL, with the range being 8.01 fL to 10.00 fL (62.5%). Patients in group B had a median MPV of 10.65 fL (range: 10.01-12.00 fL; n=24; 66.67%). Most patients (n=22, 55%) in the control group had an MPV of 8.01 fL to 10.00 fL. (mean: 7.98 fL).

Table 13: FBS Vs MPV

Pearson's "r"Correlation	Multiple R	R Square	P Value
FBS Vs MPV	0.561742	0.315554	<0.0001

When we compared the FBS levels of our study group to the MPV levels, we found a positive correlation (pearson's coefficient = 0.5617, p <0.0001).

Table 14: PPBS Vs MPV

Pearson's "r" Correlation	Multiple R	R Square	P Value
PPBS Vs MPV	0.522518	0.273025	<0.0001

Our research found a positive Pearson's coefficient of 0.5225 between PPBS and MPV, with a significance level of p <0.0001.

Table 15: HBA1c Vs MPV

Pearson's "r" Correlation	Multiple R	R Square	P Value
HBA1c Vs MPV	0.64108	0.494552	<0.0001

With a pearson's coefficient of 0.6410, the degree of correlation between HBA1c and MPV in our study participants was statistically significant (p <0.0001).

DISCUSSION

According to the findings of our research, we discovered that the FBS was significantly higher in group B in comparison to group A, with a mean difference of 36.38 mg/dl. On the other hand, the FBS was found to be higher in group A in comparison to the control group, with a mean difference of 28.90 mg/dl; however, it was found to be significantly higher in group B in comparison to the control group, with a mean difference of 65.28 mg/dl (42 percent). The results of an unpaired t-test indicated that there were significant differences between the groups. (p <0.0001).[11-13]

According to the findings of our research, the participants in Group B had PPBS levels that were 49 percent higher than those of the control group, while the participants in Group A had PPBS levels that were 34 percent higher than those of the control group, and the participants in Group B had PPBS levels that were 22 percent higher than those of the control group. The results of a statistical test called an unpaired t-test indicated that differences were statistically significant at the levels of p=0.0019, <0.0001, and <0.0001 correspondingly.[13-15]

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

Platelet counts per litre in group B were found to have increased by 6% when compared to group A's results. The difference in platelet counts between groups A and C was 24.18 (X 10 /L), which translates to group A having a count that was 9 percentage points higher. The platelet counts of Group B were 40.98 (X 10 /L) higher than the platelet counts of the control group, which means that Group B had 14% more platelets. After doing a three-way unpaired t-test, the following p-values were found to be significant: 0.0185, <0.0001, and <0.0001.[15-17]

When we compared groups A and B, we discovered that the platelet count in group B was significantly higher than that of group A by 16.80 (X 10 /L), which is a difference of 6%. On the other hand, the platelet count in group A was significantly higher than that of the control group by 24.18 (X 10 /L), which is a difference of 9%, and the platelet count in group B was significantly higher than that of the control group by 40.98 (X 10 / After maintaining a consistent level of mean platelet volume across all of the research groups, there was a 16% rise in group B's platelet count when compared to group C's results. The possibility of something happening is extremely low given that the unpaired t-p value test's result was less than <0.0001, as expected.[17-19]

According to the correlation study, MPV concentrations had a high link with both fasting blood sugar and postprandial blood sugar levels. They also had a substantial association with haemoglobin A1c levels. Although there is a linear association between an increase in FBS and an increase in MPV level 56% of the time, this variation is only managed effectively 32% of the time. This is despite the fact that this relationship holds true 56% of the time. The linear increase in MPV level measurement that occurs in proportion to an increase in PPBS is only correctly managed 27% of the time, despite the fact that this volatility is right 52% of the time.[19,20]

CONCLUSION

In this study, both well-controlled and poorly-controlled diabetics had significantly higher fasting blood sugar levels compared to healthy controls. In contrast to people whose diabetes was under control, uncontrolled diabetic patients had far higher FBS levels. Uncontrolled diabetes was associated with 1.31 times higher FBS levels than controlled diabetes, 1.32 times higher FBS levels than diabetics, and 1.73 times higher FBS levels than non-diabetics.

Patients with both well-controlled and poorly-controlled diabetes exhibited substantially higher PPBS levels than healthy controls. Patients whose diabetes was not under control had much higher FBS levels. When comparing those with controlled and uncontrolled diabetes, as well as those with diabetes and those without diabetes, the PPBS levels were 1.29, 1.52, and 1.96 times higher, respectively. Both well-controlled and poorly-controlled diabetics exhibited greater platelet counts than non-diabetic patients. Platelet counts are higher in patients whose diabetes is not under control. This means that the number of platelets in the blood of those with uncontrolled diabetes was 1.06 times higher than in those with controlled diabetes, 1.10 times higher than in non-diabetics, and 1.17 times higher than in non-diabetics.

Subjects with both controlled and uncontrolled diabetes exhibited significantly larger mean platelet volume compared to non-diabetic patients. Patients whose diabetes is not under control have a significantly increased platelet count. That is to say, people with uncontrolled diabetes had 1.12 times more platelets than those with controlled diabetes, 1.58 times more than non-diabetics, and 1.68 times more than non-diabetics. 5% linear increases in FBS, PPBs, and HbA1c were linked with increases in MPV levels.

References

1. Ismail, N., Becker, B., Strzelczyk, P., & Ritz, E. (1999). Renal disease and hypertension in non–insulin-dependent diabetes mellitus. Kidney international, 55(1), 1-28.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

2. Parving, H. H., Hommel, E., Mathiesen, E., Skøtt, P., Edsberg, B., Bahnsen, M., ... & Lauritzen, E. (1988). Prevalence of microalbuminuria, arterial hypertension, retinopathy, and neuropathy in patients with insulin dependent diabetes. Br Med J (Clin Res Ed), 296(6616), 156-160.
3. Standl, E., &Stiegler, H. (1993). Microalbuminuria in a random cohort of recently diagnosed type 2 (non-insulin-dependent) diabetic patients living in the greater Munich area. Diabetologia, 36(10), 1017-1020.
4. Schmitz, A., Vaeth, M., &Mogensen, C. E. (1994). Systolic blood pressure relates to the rate of progression of albuminuria in NIDDM. Diabetologia, 37(12), 1251-1258.
5. Zimmet, P., Alberti, K. G. M. M., & Shaw, J. (2001). Global and societal implications of the diabetes epidemic. Nature, 414(6865), 782-787.
6. Braunwald, E., Fauci, A. S., Hauser, S. L., Longo, D. L., & Jameson, J. L. (2005). Harrison's principles of internal medicine. McGraw-Hill Companies, Inc.
7. Kronenberg, H. M., ShlomoMelmed, M. D., Polonsky, K. S., Wilson, J. D., Foster, D. W., &Kronenberg, H. M. (2002). Williams textbook of endocrinology.
8. Papanas, N., Symeonidis, G., Maltezos, E., Mavridis, G., Karavageli, E., Vosnakidis, T. H., & Lakasas, G. (2004). Mean platelet volume in patients with type 2 diabetes mellitus. Platelets, 15(8), 475-478.
9. Valeri, C. R., Feingold, H., Cassidy, G., Ragno, G., Khuri, S., &Altschule, M. D. (1987). Hypothermia-induced reversible platelet dysfunction. Annals of surgery, 205(2), 175.
10. Gladwin, A. M., & Martin, J. F. (1990). The control of megakaryocyte ploidy and platelet production: biology and pathology. The International Journal of Cell Cloning, 8(4), 291-298.
11. O'malley, T., Langhorne, P., Elton, R. A., & Stewart, C. (1995). Platelet size in stroke patients. Stroke, 26(6), 995-999.
12. Jakubowski, J. A., Thompson, C. B., Vaillancourt, R., Valeri, C. R., &Deykin, D. (1983). Arachidonic acid metabolism by platelets of differing size. British journal of haematology, 53(3), 503-511.
13. Frojmovic, M. M., & Milton, J. G. (1982). Human platelet size, shape, and related functions in health and disease. Physiological reviews, 62(1), 185-261.
14. Thompson, C. B., & Jakubowski, J. A. (1988). The pathophysiology and clinical relevance of platelet heterogeneity.
15. Vizioli, L., Muscari, S., & Muscari, A. (2009). The relationship of mean platelet volume with the risk and prognosis of cardiovascular diseases. International journal of clinical practice, 63(10), 1509-1515.
16. Trowbridge, E. A. (1990). The circulating megakaryocyte, platelet volume heterogeneity and thrombopoiesis. In Platelet Heterogeneity (pp. 155-183). Springer, London.
17. Suvorov, A. V., &Markosyan, R. A. (1981). Some mechanisms of the effect of EDTA on platelet aggregation. Bulletin of Experimental Biology and Medicine, 91(5), 651-653.
18. Endler, G., Klimesch, A., Sunder-Plassmann, H., Schillinger, M., Exner, M., Mannhalter, C., ... & Sunder-Plassmann, R. (2002). Mean platelet volume is an independent risk factor for myocardial infarction but not for coronary artery disease. British journal of haematology, 117(2), 399-404.
19. Sharpe, P. C., Desai, Z. R., & Morris, T. C. (1994). Increase in mean platelet volume in patients with chronic renal failure treated with erythropoietin. Journal of clinical pathology, 47(2), 159-161.
20. Jagroop, I. A., Tsiara, S., & Mikhailidis, D. P. (2003). Mean platelet volume as an indicator of platelet activation: methodological issues. Platelets, 14(5), 335-336.