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Comparison Of Mean Platelet Volume In Patients With Type-2 Diabetes Mellitus On Insulin Therapy And On Oral Hypoglycemic Agents.

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Abstract

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder associated with persistent hyperglycemia and a high risk of microvascular complications, particularly diabetic retinopathy. Platelet dysfunction and increased platelet activation play a significant role in the pathogenesis of vascular complications. Mean platelet volume (MPV), an indicator of platelet size and activity, has emerged as a potential biomarker reflecting platelet reactivity and thrombotic risk. ***Objectives:*** To compare mean platelet volume in patients with T2DM receiving insulin therapy and those on oral hypoglycemic agents Methods: A cross-sectional observational study was conducted over 18 months at a tertiary care hospital. A total of 100 patients with confirmed T2DM were enrolled, comprising 50 patients on insulin therapy and 50 on oral hypoglycemic agents. Patients with confounding conditions affecting platelet function were excluded. Data collection included clinical evaluation, laboratory investigations (MPV, fasting and postprandial blood glucose, HbA1c, complete blood count), and fundoscopic examination. Statistical analysis was performed using SPSS, with significance set at $p < 0.05$. ***Results:*** The study population showed comparable distribution across age groups and smoking status between treatment modalities, minimizing confounding effects. MPV, as a marker of platelet activation, demonstrated relevance in assessing vascular risk. Elevated MPV was associated with enhanced platelet reactivity and microvascular dysfunction. The findings support the role of platelet activation in the pathogenesis of diabetic retinopathy and suggest that differences in metabolic control and treatment modality may influence platelet behavior. ***Conclusion:*** Mean platelet volume is a simple, cost-effective, and readily available parameter that reflects platelet activation in T2DM. It may serve as a useful adjunct in evaluating microvascular risk and correlating with diabetic retinopathy. Incorporating MPV into routine clinical assessment can aid in early identification of patients at higher risk of complications.

Keywords: Type 2 diabetes mellitus, Mean platelet volume, Diabetic retinopathy, Platelet activation, Insulin therapy, Oral hypoglycemic agents, Microvascular complications.

INTRODUCTION

Type 2 diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycemia resulting from a combination of insulin resistance and relative insulin deficiency. It is one of the most prevalent non-communicable diseases worldwide and represents a major public health concern due to its progressive nature and wide range of systemic complications. The burden of type 2 diabetes mellitus continues to increase globally, particularly in developing countries, driven by urbanization, sedentary lifestyle, dietary changes, obesity, and aging populations. The chronic hyperglycemic state associated with diabetes leads to metabolic, biochemical, and structural alterations in multiple organ systems, resulting in both microvascular and macrovascular complications that significantly contribute to morbidity, mortality, and reduced quality of life [1].

Among the complications of type 2 diabetes mellitus, microvascular complications such as diabetic retinopathy, nephropathy, and neuropathy play a crucial role in long-term disability.

Platelets play a central role in hemostasis and thrombosis and are increasingly recognized as active participants in inflammatory and atherothrombotic processes. Beyond their traditional role in clot formation, platelets release a variety of

bioactive substances, including growth factors, cytokines, and pro-inflammatory mediators, which contribute to endothelial dysfunction and vascular injury.

Mean platelet volume is a routinely measured hematological parameter that reflects the average size of circulating platelets and serves as an indirect marker of platelet activation and reactivity. Larger platelets are metabolically and enzymatically more active, contain more dense granules, and have greater prothrombotic potential compared to smaller platelets.

Assessing mean platelet volume in patients with type 2 diabetes mellitus receiving different therapeutic modalities may contribute to a better understanding of platelet-mediated mechanisms in diabetic microvascular disease.

MATERIALS AND METHODS

This cross-sectional observational study was conducted in the Department of General Medicine, Krishna Rajendra Hospital, Mysore Medical College and Research Institute (MMC & RI), Mysore. Patients attending the general medicine outpatient department as well as those admitted to the wards were screened for eligibility. The study was conducted over a period of 18 months, from April 2024 to September 2025. Ethical approval for the study was obtained from the Institutional Ethics Committee of Mysore Medical College and Research Institute, Mysore, prior to the commencement of the study.

Inclusion Criteria

1. Patients with confirmed Type 2 Diabetes Mellitus who were on insulin therapy for a minimum duration of six months.
2. Patients with confirmed Type 2 Diabetes Mellitus who were on oral hypoglycemic agents for a minimum duration of six months.

Exclusion Criteria

1. Patients with Type 1 Diabetes Mellitus.
2. Patients with abnormal platelet count or abnormal platelet morphology.
3. Patients receiving antiplatelet medications.
4. Patients diagnosed with chronic kidney disease.
5. Patients with chronic liver disease.
6. Patients receiving both insulin and oral hypoglycemic agents.
7. Patients with myelodysplastic syndrome.
8. Patients with myeloproliferative disorders.
9. Patients with inflammatory bowel disease.
10. Patients with functional platelet disorders.
11. Patients who had undergone splenectomy.
12. Patients with thyroid disorders.
13. Patients with splenomegaly.

Study Sampling

Sampling was done using a convenience sampling method. Eligible patients fulfilling the inclusion criteria and willing to participate were enrolled consecutively until the required sample size was achieved. Selection of participants was based on their availability during the study period in the outpatient and inpatient settings.

Study Sample Size

The sample size was calculated using the following formula: $S= Z^2 PQ/D^2$
Where:

- S = Sample size
- Z = Standard normal deviate at 95% confidence level (1.96)
- P = Estimated prevalence of diabetes (10% or 0.10)
- Q = 1 − P = 0.90
- D = Margin of error (6% or 0.06)

The calculated sample size was 96, which was rounded off to 100 participants for the study.

Study Parameters

The following parameters were assessed in all study participants:

- Mean platelet volume
- Fasting blood sugar
- Postprandial blood sugar
- Glycated hemoglobin (HbA1c)

- Urine analysis for albumin and sugar
- Complete blood count, including platelet count
- Blood urea
- Serum creatinine
- Fundoscopic findings

Study Procedure

After obtaining approval from the Institutional Ethics Committee, the study was initiated in the Department of General Medicine. Eligible patients were identified during their visit to the outpatient department or during hospital admission and were screened based on the predefined inclusion and exclusion criteria. The purpose and details of the study were explained to the patients and their attendants, and written informed consent was obtained prior to participation.

A detailed clinical history was recorded for each participant, followed by a thorough general physical and systemic examination. Blood samples were collected under aseptic precautions for laboratory investigations, including mean platelet volume, fasting blood sugar, postprandial blood sugar, HbA1c, complete blood count, blood urea, and serum creatinine. Urine samples were obtained for routine analysis of albumin and sugar. All participants underwent fundoscopic examination to assess retinal changes. The findings from clinical evaluation and investigations were documented systematically in a pre-designed proforma for further analysis.

Study Data Collection

Data were collected using a pre-designed and structured proforma for all enrolled participants. The proforma included demographic details, relevant clinical history, findings of physical and systemic examination, and results of laboratory and fundoscopic investigations. All laboratory values were recorded from reports obtained during the study period, and fundoscopic findings were documented after examination.

The collected data were entered systematically to ensure completeness and accuracy. Each participant was assigned a unique identification number to maintain confidentiality. Data collection was carried out consistently throughout the study period to ensure uniformity and minimize recording errors.

Statistical Analysis

Data collected were entered into Microsoft Excel and analyzed using SPSS software (trial version). Descriptive statistics were expressed in the form of mean and standard deviation for continuous variables, and frequencies, percentages, and proportions for categorical variables. The association between categorical variables among the two groups was analyzed using the Chi-square test. A p-value less than 0.05 was considered to indicate a statistically significant association between the groups and the categorical variables. Differences between the two groups with respect to continuous variables were analyzed using the one-sample t-test, and a p-value less than 0.05 was considered to denote a statistically significant difference between the mean values of the groups.

RESULTS

Table 1. Association between age group and treatment modality

Age group (years)	Insulin n (%)	OHA n (%)	Total
40–55	25 (50.0)	22 (44.0)	47
56–70	25 (50.0)	28 (56.0)	53
Total	50	50	100
Chi-square = 0.361, df = 1, p = 0.548			

There was no statistically significant association between age group and treatment modality in the study population. Both insulin and oral hypoglycemic groups showed comparable age distribution. Approximately half of the patients in each group belonged to both age categories. This indicates that age was evenly distributed between treatment groups. Hence, age is unlikely to act as a confounding factor while comparing outcome variables. The two groups were well matched with respect to age. Smoking status did not show a statistically significant association with treatment modality. Both groups had a comparable proportion of smokers and non-smokers. This suggests that smoking habits were evenly distributed among insulin and oral hypoglycemic groups. Smoking is therefore unlikely to influence intergroup differences observed in platelet or glycemic parameters. The groups were comparable with respect to smoking status.

Alcohol consumption was not significantly associated with treatment modality. Both insulin and oral hypoglycemic groups had similar proportions of alcohol consumers. The absence of statistical significance indicates balanced distribution of alcohol intake across groups. Therefore, alcohol consumption is unlikely to confound the study outcomes. The baseline comparability of lifestyle factors was maintained.

No statistically significant association was observed between urine albumin status and treatment modality. The proportion of patients with albuminuria was similar in both groups. This suggests comparable renal involvement among insulin and oral hypoglycemic groups. Hence, renal status was balanced at baseline. Urine albumin is unlikely to bias the observed associations in the study.

Urine sugar distribution was identical in both insulin and oral hypoglycemic groups. There was no statistically significant association between urine sugar status and treatment modality. This reflects uniform glycosuria patterns across groups at baseline. The identical distribution further confirms excellent group comparability. Urine sugar is unlikely to influence intergroup outcome differences.

Table 2. Association between hypertension and treatment modality

Hypertension	Insulin n (%)	OHA n (%)	Total
No	18 (36.0)	26 (52.0)	44
Yes	32 (64.0)	24 (48.0)	56
Total	50	50	100
Chi-square = 2.597, df = 1, p = 0.107			

Hypertension was not significantly associated with treatment modality. Both groups had a substantial proportion of hypertensive patients. The difference observed was not statistically significant. This suggests comparable cardiovascular risk profiles across groups. Hypertension is unlikely to confound the primary outcomes of the study.

Dyslipidemia did not show a statistically significant association with treatment modality. The prevalence of dyslipidemia was similar in both insulin and oral hypoglycemic groups. This indicates balanced lipid profiles at baseline. Dyslipidemia is therefore unlikely to influence observed differences in platelet or retinal outcomes. Baseline cardiovascular risk factors were well matched.

Table 3. Baseline demographic and treatment-related variables according to treatment group

Variable	Insulin (n=50) Mean ± SD	OHA (n=50) Mean ± SD	t-value	p-value
Age (years)	56.40 ± 9.36	57.24 ± 8.86	–0.461	0.646
Duration of treatment (months)	36.06 ± 14.71	32.32 ± 16.40	1.200	0.233
BMI (kg/m²)	25.34 ± 2.52	25.36 ± 2.26	–0.042	0.967

The baseline demographic and treatment-related characteristics were comparable between the insulin and oral hypoglycemic groups. There was no statistically significant difference in age, duration of treatment, or body mass index between the two groups. This indicates that both groups were well matched at baseline. The absence of significant differences suggests minimal selection bias. These comparable baseline characteristics allow for valid comparison of biochemical and platelet parameters between groups.

Table 4. Comparison of hemodynamic and glycemic parameters between Insulin and OHA groups

Variable	Insulin (n=50) Mean ± SD	OHA (n=50) Mean ± SD	t-value	p-value
Systolic BP (mmHg)	136.18 ± 14.74	130.18 ± 15.05	2.014	0.047
Diastolic BP (mmHg)	83.18 ± 7.90	85.58 ± 9.16	–1.403	0.164
Fasting blood sugar (mg/dL)	115.92 ± 14.94	178.20 ± 22.09	–16.513	<0.001
Postprandial blood sugar (mg/dL)	173.84 ± 15.20	253.52 ± 32.36	–15.760	
HbA1c (%)	7.04 ± 0.29	8.87 ± 0.94	–13.217	

Glycemic parameters were significantly higher in patients receiving oral hypoglycemic agents compared to those on insulin therapy. Both fasting and postprandial blood glucose levels showed highly significant differences between the groups. HbA1c was also significantly elevated in the oral hypoglycemic group, indicating poorer long-term glycemic control. Systolic blood pressure was marginally higher in the insulin group, while diastolic blood pressure showed no significant difference. Overall, insulin therapy was associated with superior glycemic control.

Table 5. Comparison of platelet and renal parameters between Insulin and OHA groups

Variable	Insulin (n=50) Mean ± SD	OHA (n=50) Mean ± SD	t-value	p-value
Platelet count (×10³/µL)	282.86 ± 54.79	294.38 ± 60.96	–0.994	0.323
Mean platelet volume (fL)	7.52 ± 0.15	8.57 ± 0.33	–20.832	<0.001
Blood urea (mg/dL)	30.68 ± 5.95	30.56 ± 5.87	0.101	0.919
Serum creatinine (mg/dL)	0.95 ± 0.16	0.93 ± 0.15	0.503	0.616

Mean platelet volume was significantly higher in patients treated with oral hypoglycemic agents compared to those on insulin therapy. This difference was highly statistically significant, indicating increased platelet activation in the oral hypoglycemic group. Platelet count did not differ significantly between groups, suggesting that platelet size rather than number was affected. Renal parameters, including blood urea and serum creatinine, were comparable between the two groups. These findings support MPV as a sensitive marker of platelet activation independent of renal function.

DISCUSSION

By comparing patients on insulin therapy with those on oral hypoglycemic agents, the study sought to identify whether treatment-related differences in glycemic control were associated with corresponding differences in mean platelet volume and retinal status. The significance of the study is further enhanced by the practical applicability of mean platelet volume as a low-cost and easily accessible laboratory marker, particularly in resource-limited settings where advanced screening tools may not always be feasible. If elevated mean platelet volume is consistently associated with poor glycemic control and diabetic retinopathy, it may serve as an adjunctive marker for identifying patients at higher risk of microvascular complications.

Age group and treatment modality

The present study demonstrated no statistically significant association between age group and treatment modality, indicating that the insulin and oral hypoglycemic agent (OHA) groups were well matched with respect to age. The mean age also remained comparable, being 56.40 ± 9.36 years in the insulin group and 57.24 ± 8.86 years in the OHA group (p = 0.646). This balanced age distribution is important because advancing age is a recognized risk factor for longer disease exposure, poorer metabolic status, and greater vascular complication burden in type 2 diabetes mellitus. A similar age pattern has been reported in previous studies.

Tasneem et al. observed a mean age of 53.3 years in diabetic patients assessed for retinopathy, indicating that platelet activation and retinal complications are largely concentrated in middle-aged and older individuals [2]. In contrast to those studies, which mainly highlighted age as a contributor to complication prevalence, the present study specifically established that age distribution was similar in both treatment groups. This strengthens the internal validity of the comparison because the subsequent differences observed in glycemic status, MPV, and diabetic retinopathy are less likely to be attributable to age imbalance. Thus, age did not appear to act as a confounding variable in the present analysis and the observed intergroup differences may be interpreted with greater confidence.

Smoking status and treatment modality

Smoking status did not show a statistically significant association with treatment modality in the present study, suggesting that tobacco exposure was evenly distributed between insulin-treated and OHA-treated patients. This comparability is relevant because smoking is known to aggravate endothelial dysfunction, oxidative stress, platelet activation, and vascular inflammation, all of which may worsen diabetic microvascular complications including retinopathy. Although the studies provided for comparison did not present subgroup analyses specifically based on smoking habits, they consistently emphasized the relationship between platelet activation, poor glycemic control, and microvascular complications.

Kodiatte et al. showed that MPV was significantly elevated in diabetic patients and positively correlated with fasting blood glucose, postprandial glucose, and HbA1c, supporting the role of metabolic derangement in platelet activation rather than isolated lifestyle variables [3]. In contrast to those studies, which were primarily designed to explore platelet indices and retinal complications, the present study adds methodological strength by demonstrating balanced smoking distribution between treatment groups. Therefore, the significantly higher MPV and greater prevalence of diabetic retinopathy in the OHA group are less likely to be explained by unequal smoking exposure. Instead, the findings more strongly support an association with poorer glycemic control in that group. Thus, smoking status was not a major confounding factor in the present comparison and the observed treatment-related differences appear to reflect true variation in metabolic and microvascular burden.

Alcohol consumption and treatment modality

The present study found no statistically significant association between alcohol consumption and treatment modality, indicating that alcohol-related lifestyle exposure was broadly comparable between the insulin and OHA groups. Such baseline comparability is important because alcohol may influence metabolic control, hepatic function, vascular health, and platelet activity depending on the amount and duration of consumption. However, the absence of significant imbalance in the present study suggests that alcohol use is unlikely to explain the higher MPV levels and greater frequency of diabetic retinopathy observed in the OHA group.

The comparative studies provided by Kodiatte et al., Dindar et al., Tuzcu et al., and Reddy et al. did not furnish direct subgroup analyses according to alcohol consumption, but their findings consistently linked platelet activation more strongly to hyperglycemia and vascular complications than to lifestyle descriptors alone [3, 5, 6]. For instance, Kodiatte et al. demonstrated a significant association between MPV and glycemic variables such as fasting blood glucose, postprandial glucose, and HbA1c [3], while Dindar et al. found that higher MPV was associated with poor glycemic control and retinopathy [4]. In contrast to those studies, the present work explicitly confirmed that alcohol consumption was not differentially distributed between treatment groups. This strengthens the inference that the significant intergroup differences in glycemic parameters, MPV, and retinal findings are more plausibly attributable to metabolic status and treatment profile rather than unequal alcohol exposure.

Urine albumin and treatment modality

The present study showed no statistically significant association between urine albumin status and treatment modality, indicating comparable renal microvascular involvement across the insulin and OHA groups. Since albuminuria is an indicator of diabetic nephropathy and generalized endothelial dysfunction, balanced distribution of urine albumin status between groups is methodologically important. It suggests that renal microvascular burden was similar at baseline and therefore unlikely to account for the differences noted later in MPV and diabetic retinopathy. Dindar et al. assessed diabetic microvascular complications collectively and found that MPV was significantly higher in patients with such complications, especially in those with poor glycemic control and retinopathy [4].

These studies support the broader concept that platelet activation is linked to microvascular disease burden. In contrast to those studies, which were centered on complication status rather than treatment-group baseline balance, the present study specifically established that albuminuria did not differ significantly between insulin-treated and OHA-treated patients. This finding is important because it reduces the likelihood that unequal nephropathy burden influenced the observed association of higher MPV and greater retinopathy prevalence in the OHA group. Thus, the significant retinal and platelet differences seen in the present study are better interpreted as being related to poorer glycemic status and treatment profile rather than to disproportionate renal involvement. Therefore, urine albumin did not function as a major confounding factor and the internal validity of the intergroup comparison remains strong.

Urine sugar and treatment modality

Urine sugar distribution was identical in both treatment groups in the present study, demonstrating excellent baseline comparability with respect to glycosuria. However, despite this identical distribution, major differences were observed in fasting blood glucose, postprandial blood glucose, and HbA1c, suggesting that urine sugar is a relatively crude marker and may not accurately capture the full extent of glycemic burden. This interpretation is strongly supported by Kodiatte et al., who showed that MPV correlated significantly with fasting blood glucose, postprandial blood glucose, and HbA1c ($p = 0.001$), establishing that direct biochemical indices of glycemic control are more informative in understanding platelet activation than indirect markers alone [3].

Dindar et al. similarly demonstrated significant positive associations of MPV with fasting blood glucose ($p = 0.03$) and HbA1c ($p < 0.001$), indicating that chronic and sustained hyperglycemia rather than isolated urinary findings is more relevant to platelet reactivity [4]. In the present study, although urine sugar status was exactly matched between treatment groups, the OHA group showed markedly higher fasting blood sugar (178.20 ± 22.09 mg/dL vs 115.92 ± 14.94 mg/dL), postprandial blood sugar (253.52 ± 32.36 mg/dL vs 173.84 ± 15.20 mg/dL), and HbA1c (8.87 ± 0.94% vs 7.04 ± 0.29%).

Hypertension and treatment modality

Hypertension did not show a statistically significant association with treatment modality in the present study, although it was relatively common in both groups.

Although numerically more patients in the insulin group were hypertensive, the difference did not reach statistical significance, indicating that blood pressure status was reasonably comparable between the two treatment arms. This is relevant because hypertension is an established risk factor for endothelial dysfunction and progression of diabetic retinopathy. Therefore, balanced distribution reduces the likelihood that the observed differences in retinal findings and

MPV were driven primarily by unequal hypertensive burden. Dindar et al. included blood pressure measurements in their analysis of MPV and diabetic microvascular complications, underscoring that vascular risk must be interpreted in the context of multiple contributing factors [4].

In contrast to those studies, which mainly examined complication status across diabetic patients, the present study specifically confirmed that hypertension itself was not significantly different between insulin-treated and OHA-treated individuals. This enhances confidence in the interpretation that the higher MPV and greater prevalence of retinopathy in the OHA group are more strongly related to poorer glycemic control than to differences in hypertensive status. Thus, while hypertension remains clinically important in diabetic vascular disease, it did not appear to be a major confounder of treatment-group comparison in the present study.

Baseline demographic and treatment-related variables according to treatment group

The present study demonstrated that baseline demographic and treatment-related variables were comparable between the insulin and OHA groups, thereby strengthening the validity of subsequent intergroup comparisons. Findings confirm that the two groups were well matched with respect to age, treatment duration, and body mass index, reducing the likelihood of baseline selection bias. Such comparability is important because age, obesity, and chronic disease exposure are known contributors to insulin resistance, poor glycemic control, platelet activation, and vascular complications. Kodiatte et al. similarly assessed MPV in relation to body mass index and duration of diabetes while studying type 2 diabetic patients, emphasizing that these baseline clinical factors are relevant in interpreting platelet behavior [3].

In contrast to studies where baseline heterogeneity may have influenced vascular outcomes, the present study clearly established that these variables were balanced between the insulin and OHA groups. Therefore, the significant differences observed later in glycemic parameters, MPV, and diabetic retinopathy are less likely to be due to unequal age, disease duration, or body habitus, and more likely to represent true treatment-group-associated differences in metabolic and vascular burden.

Hemodynamic and glycemic parameters between insulin and OHA groups

The present study revealed marked differences in glycemic parameters between the insulin and OHA groups, while hemodynamic differences were limited. Findings indicate far better short-term and long-term glycemic control in the insulin group. Kodiatte et al. reported that MPV correlated significantly with fasting blood glucose, postprandial blood glucose, and HbA1c, all with $p = 0.001$, indicating that worsening glycemic control is closely associated with platelet activation [3]. Dindar et al. similarly demonstrated positive associations between MPV and fasting blood glucose ($p = 0.03$) and HbA1c ($p < 0.001$) [4]. In contrast to those studies, the present study shows that treatment modality itself was associated with a profound glycemic difference, which likely underlies the lower MPV and lower retinopathy burden observed among insulin-treated patients.

Platelet and renal parameters between insulin and OHA groups

The present study demonstrated that platelet count and renal parameters were comparable between insulin-treated and OHA-treated patients, whereas mean platelet volume differed markedly. In contrast, MPV showed a highly significant difference, being 7.52 ± 0.15 fL in insulin-treated patients and 8.57 ± 0.33 fL in OHA-treated patients ($t = –20.832$, $p < 0.001$). These findings indicate that platelet size and activity rather than platelet number differed according to treatment modality, and that renal function was unlikely to explain the observed difference in MPV.

Kodiatte et al. reported a similar pattern, finding that platelet counts were only slightly higher in diabetic patients than in non-diabetic controls and not statistically significant ($277.46 \pm 81 \times 10^9$/L vs $269.79 \pm 78 \times 10^9$/L; $p = 0.256$), whereas MPV was significantly increased (8.29 ± 0.74 fL vs 7.47 ± 0.73 fL; $p = 0.001$) [56]. Dindar et al. also found a significant negative correlation between MPV and platelet count ($p < 0.001$), supporting the view that larger platelets may reflect accelerated platelet turnover rather than increased platelet number [4]. In contrast to the stability of platelet count and renal indices, the striking rise in MPV in the OHA group of the present study supports MPV as a more sensitive and clinically meaningful marker of diabetic vascular risk.

CONCLUSION

The findings clearly demonstrated that both treatment groups were broadly comparable with respect to baseline demographic variables, lifestyle factors, urinary findings, body mass index, duration of treatment, renal function, hypertension, and dyslipidemia. A major conclusion emerging from the study is that glycemic control was significantly superior in the insulin-treated group. The study further establishes that mean platelet volume is closely associated with treatment modality and metabolic control.

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