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EVALUATION OF HEMATOLOGICAL CHANGES IN COMPLICATED DIABETES: A PILOT STUDY

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ABSTRACT

Background: Diabetes mellitus is a chronic metabolic disorder frequently complicated by microvascular and macrovascular manifestations. Alterations in hematological parameters are increasingly recognized as potential indicators of disease progression and risk of complications. This study investigates the association between routine hematological indices and the presence of complications in patients with type 2 diabetes mellitus (T2DM).

Materials and Methods: A hospital-based cross-sectional pilot study was conducted at Madha Medical College, enrolling 24 adults with confirmed T2DM. Participants were divided into two groups: those with diabetes-related complications (n=12) and those without complications (n=12). Demographic, clinical, and laboratory data including white blood cell (WBC), red blood cell (RBC), and platelet indices were collected and analyzed. Group comparisons employed independent samples t-tests, with $p<0.05$ considered statistically significant.

Results: The complication group exhibited significantly higher hemoglobin (11.95 ± 1.82 g/dL vs. 9.98 ± 2.19 g/dL, $p=0.026$) and hematocrit ($35.76 \pm 5.54\%$ vs. $29.63 \pm 7.31\%$, $p=0.030$). WBC counts trended lower in the complication group ($p=0.056$). Platelet indices showed no significant intergroup differences, though subtle shifts were noted.

Conclusion: Significant elevations in hemoglobin and hematocrit were identified among patients with diabetes-related complications, while other hematological indices demonstrated suggestive, but non-significant, trends. These findings imply that standard hematological parameters may provide supplementary information for identifying T2DM patients at increased risk for complications. Larger, longitudinal studies are warranted to validate these pilot findings and explore their clinical utility.

Keywords: Diabetic complications, Hematology, RBC parameters, WBC parameters, Platelet parameters, Hemoglobin, Hematocrit, PDW.

INTRODUCTION

Diabetes Mellitus is a chronic condition associated with elevated blood glucose levels due to either reduced production of the hormone insulin by the body, or the ineffective utilization of the produced insulin by the body.[1] It is considered to be a heterogenous group of metabolic disorders marked by defects in carbohydrate, fat or protein metabolism.[2] Diabetes is characterized by hyperglycemia and hyperinsulinemia, often associated with insulin resistance in peripheral tissues.[3] Diabetes mellitus is the major global epidemic of the present day, with 589 million adults aged 20-79 years living with diabetes, which accounts to 1 in 9 adults worldwide.[4] The prevalence of diabetes mellitus patients is predicted to be 592 million by the year of 2035, according to the World Health Organization (WHO).[5] Diabetes has steadily increased in India and around the world over the last three decades, with India accounting for a sizable portion of the global burden. India has the second highest number of adults in the world - 89.8 million diabetics . Apart from the rising prevalence of diabetes in India, the incidence of diabetes is also

rising steadily, with a fast transition from euglycemia to prediabetes and diabetes.[6] Diabetes is one of the largest global health emergencies of this century, ranking among the 10 leading causes of mortality together with cardiovascular disease (CVD), respiratory disease, and cancer.[7] According to the World Health Organization (WHO), Diabetes was among the top 10 causes of death in India, with a significant 19.55 deaths per 100,000 population. Glycaemic imbalance is merely one aspect of the burden of diabetes; chronic exposure to high glucose levels progressively damages several organ systems, leading to microvascular and macrovascular problems. In addition to its effects on systemic metabolism, diabetes mellitus's chronic hyperglycemia triggers a series of molecular and cellular changes that have a major effect on blood components, such as red blood cells, white blood cells, and platelets, and thereby exacerbate the microvascular and macrovascular complications of the disease.

Prolonged hyperglycemia causes non enzymatic interaction between the carbonyl group of the reducing sugar and the primary amino group of a protein, the end product of this reaction being termed advanced glycation end products (AGE).The molecules associated with AGE change into oxidants and acquire distinct properties. This results in the generation of reactive oxygen species (ROS), which raises oxidative stress and halts the release of nitric oxide (NO). Additionally, AGE reduces the bio-availability and activity of endothelium derived nitric oxide.[8] Such effects of AGE bring about changes in various components of blood such as Red Blood Cells, White blood Cells, platelets and their associated parameters. Hyperglycemia causes reduced red cell Na/K-ATPase activity, oxidation-induced protein structure changes, and the buildup of AGEs on the red cell membrane, all contributing to alterations in the lipid composition and deformability of the red cell membrane, as well as decreased filterability and increased adhesion of RBCs. In platelets, AGE promotes the thrombogenic state by externalising platelet membrane phosphatidylserine, which activates surface clotting factor.[8] Additional effects of hyperglycemia involve the release of larger platelets with more GPIb and GPIIb/IIIa receptors and an enhanced capacity to form thromboxane, as well as the activation of protein kinase C, a transduction pathway mediator for many proaggregatory platelet agonists.[8] Glycation of plasminogen, fibrinogen, prothrombin, and other clotting-related proteins occurs as a result of chronic hyperglycemia.[9] Plasminogen glycation reduces the catalytic efficiency of plasmin and decreases plasmin production . In addition to RBCs and platelets, white blood cells (WBCs) also exhibit numerical and functional changes in diabetes mellitus. WBC has an increased count that, even if it remains within the normal range, may be useful in predicting the severity of complications among individuals with diabetes. Furthermore, monocyte and neutrophil counts rose in tandem with the development of complications. The chemotactic, phagocytic, and bactericidal functions of neutrophils of diabetics are compromised. Myeloperoxidase activity, lysosomal enzyme release, and neutrophil ROS generation are all reduced . The vulnerability to infection gets impacted by these enzyme alterations. Decreased leukocyte-endothelial cell interactions, fewer leukocytes in inflammatory lesions, low superoxide generation, decreased leukocyte release of TNF-α, IL1ß, and prostaglandin E2 upon exposure to lipopolysaccharide, and low neutrophil arachidonic acid content could all be contributing factors to the increased severity of infections in diabetics.

RBC parameters such as hemoglobin and hematocrit are altered in patients with diabetes mellitus and can reflect the presence of complications. HbA1c is commonly used to assess long-term glycemic control, but diabetes also tends to alter the basic RBC parameters, such as hemoglobin (Hb) and Hematocrit (HCT), causing a reduction in both parameters leading to anemia. The etiology of anemia in diabetes is multifactorial, among which decreased renal function and pro-inflammatory cytokines are the most important determinants of anemia. The anemia is commonly normocytic and normochromic in nature and occurs even in patients without renal disease. In early diabetes, renal blood flow is increased, leading to hyperfiltration. Increased renal oxygen delivery (blood flow) may act to suppress EPO production in the diabetic kidney. Hyperglycemia induces metabolic and functional abnormalities in erythrocytes, causing the red cell surface to expose the aminophospholipid phosphatidylserine on the cell surface, thereby rendering red blood cells to be identified as senescent cells. In type 2 diabetes, erythrocyte caspase-3 can be activated by an extracellular oxidative environment, which hinders the maintenance of erythrocyte shape and function.[10] Sorbitol accumulation through the activated polyol pathway leads to an increase in osmotic stress.[11] These changes contribute to reduced erythrocyte survival and their removal by the reticuloendothelial system.[10] T2DM patients with a Hb level below 12 g/dl are two times more likely to develop background diabetes retinopathy than those with higher Hb levels and five times more likely to develop pre-proliferative or proliferative retinopathy. It could result from retinal hypoxia, which upregulates vascular endothelial growth factor and other genes related to capillary permeability, apoptosis, and neoangiogenesis. In diabetics without significant vascular problems, a positive link has been found between high RDW and an increased incidence of macro- and microvascular issues.[12]

Leukocytes play a pivotal role in the pathogenesis of diabetic complications. Elevated total leukocyte counts have been strongly associated with both microvascular and macrovascular complications, including increased risk of retinopathy, cardiac events, and nephropathy.[13] In particular, studies suggest that higher leukocyte counts correlate with

greater severity of diabetic retinopathy and cardiovascular disease.[13] In the context of diabetic nephropathy, leukocytes contribute directly to glomerular injury through oxidative and proteolytic damage to mesangial cells, promoting inflammation and fibrosis. The elevation in leukocyte count observed in diabetic patients can be explained by hormonal as well as metabolic pathways. Adipocytes may secrete more leptin in response to plasma cortisol and fluctuating insulin levels in renal disease, which would enhance the release of neutrophils from marrow storage and decrease their outflow from the bloodstream. Another hypothesis is that low blood insulin levels stimulate the generation of neutrophils in the bone marrow.[13] Beyond increased numbers, leukocytes in diabetes are also functionally activated by hyperglycemia-induced factors, including advanced glycation end-products (AGEs), reactive oxygen species (ROS), and inflammatory cytokines.These activated leukocytes release TNF-α, transforming growth factor-β (TGF-β), proteases, and superoxide radicals, which contribute to systemic oxidative stress. The resulting platelets. It provides an assessment of the circulating platelet mass. Platelet-large cell ratio (P-LCR) is the measure of larger platelets.[15] The platelet activation and dysfunction are reflected in the platelet indices. Chronic inflammatory condition and vascular complications increases platelet activation and turnover resulting in younger and more active platelets, which show alteration in platelet indices.[16,17,18] Previous studies have shown that Mean platelet volume (MPV) and platelet distribution width (PDW) are altered in diabetes.[16,19] These findings demonstrate the potential utility of platelet indices as biomarkers in Type 2 diabetes, helping to differentiate between patients with complications and patients without them.

Using routine haematological parameters as potential early indicators of vascular dysfunction and disease progression is becoming more popular due to the rising incidence of diabetes and its associated complications. Hematological parameters are inexpensive, practical, easily accessible and are readily estimated from complete blood counts that have been found to be related to a number of medical conditions and pathologies. The purpose of this study is to examine the changes in hematological parameters among individuals with type 2 diabetes and their correlation with complications from the disease with the aim to determine whether these parameters could be relevant as supplementary biomarkers for diabetes management.

MATERIALS AND METHODS

This hospital-based, cross-sectional comparative pilot study was conducted to investigate alterations in hematological parameters among patients with Type 2 Diabetes Mellitus (T2DM). The study compared patients with diabetes-related complications to those without complications, aiming to identify possible hematological variations associated with disease progression. The study was carried out at Madha Medical College and Research Institute between May and August 2024. The study population comprised adult patients with a confirmed diagnosis of T2DM who were attending outpatient or inpatient services at the institution.

Participants were included if they were 18 years or older, had a confirmed diagnosis of T2DM, and provided written informed consent. Patients with known hematological disorders such as anemia of other causes or leukemia, as well as those with chronic infections, autoimmune diseases, or malignancies, were excluded. Individuals receiving medications known to influence hematological indices, including corticosteroids, immunosuppressants, or chemotherapy, were also excluded to reduce confounding factors.

A total of 24 patients were enrolled in the study and divided equally into two groups. The control group (n = 12) consisted of diabetic patients without any documented complications, while the complications group (n = 12) included patients with one or more diabetes-related complications such as nephropathy, neuropathy, retinopathy, cardiovascular disease, or diabetic foot.

Data collection was carried out using a structured proforma that captured demographic details (age, sex), clinical history (duration of diabetes and type of complications), and laboratory investigations. Hematological parameters were measured using standard automated hematology analyzers in the hospital laboratory. White blood cell indices assessed included total WBC count, neutrophil percentage, absolute neutrophil count, lymphocyte percentage, and absolute lymphocyte count. Red blood cell indices included hemoglobin, hematocrit (HCT), and red cell distribution width (RDW). Platelet indices measured were platelet count, mean platelet volume (MPV), plateletcrit (PCT), platelet distribution width (PDW), platelet large cell concentration (PLCC), and platelet large cell ratio (PLCR).

Statistical analysis was performed using SPSS software (version to be specified). Continuous variables were expressed as mean ± standard deviation (SD) and standard error of mean (SEM). Group comparisons were conducted using the independent samples t-test, with Levene's test applied beforehand to verify homogeneity of variance. A p-value of <0.05 was considered statistically significant. Analysis revealed significant differences in hemoglobin ($p = 0.026$) and hematocrit ($p = 0.030$), both of which were higher in the complications group. No other parameters demonstrated statistically significant differences, though trends were noted for WBC count, neutrophil percentage, and RDW.

RESULTS

Demographic and Clinical Characteristics:

Demographic and clinical characteristics were compared between 12 diabetic patients with complications and 12 diabetic controls without complications. Population distribution by gender reveals that the control group has an equal number of men and women (50% male, 50% female). In contrast, the group with complications had a modest male predominance (76%), which may indicate that males are more likely than females to develop complications (24%) (Figure 1). The control group is predominantly composed of people under 60 years of age (58%), with the prevalent age group being 50-60 years (26%) and 70 to 80 years (26%), followed by individuals from the age group of less than 40 years (16%) and 40 to 50 years (16%). Comparatively, the majority of those who experience complications are between the ages of 60 and 70 (42%), followed by those between the ages of 50 and 60 (26%), and 70 and 80 (16%). This category consists primarily of those aged over 50 years, suggesting that those on this end of the age spectrum are more likely to suffer the burden of developing complications.[Figure 2]

Regarding Disease duration of Diabetes, 92% of control group have diabetes for less than 10 years, among which 50% of the control group had diabetes for less than 5 years, followed by 42% of control group who have diabetes for 5-10 years. The complication group however, displayed a bimodal distribution with 34% having diabetes for 5-10 years and 34% having diabetes for more than 20 years. This reflects the influence of chronicity of the disease on complication development (Figure 3). When analysing the mean HbA1C, it is relatively unchanged in males. Whereas there is a slight increase of HbA1C in Females with complications.

Glycemic parameters such as fasting blood sugar, post-prandial blood sugar and glycated hemoglobin were evaluated to assess glycemic control in both groups. Mean fasting blood sugar was higher in the complications group compared to controls. Among males, this difference was more pronounced, suggesting poorer fasting glycemic control in those with complications. In contrast, females showed only a marginal difference in mean FBS between groups (Figure 4). Trends in mean postprandial blood sugar varied by gender. Mean PPBS was marginally greater in the male population in the group with complications. It is noteworthy that the complication group's Mean PPBS levels were lower than those of the control group in females (Figure 5). Additionally, there was gender-based variation in mean HbA1C levels; the mean HbA1C was somewhat higher in males with complications than in the control group. However, the mean HbA1C values of females with complications are lesser than those of the control group (Figure 6).

Comparison of hematological data

Hematological parameters assessed includes WBC parameters - WBC count, Absolute Neutrophil count, Absolute Lymphocyte count; RBC parameters- Hemoglobin, RDW-CW, Hematocrit; and Platelet Parameters- Platelet Count, Mean Platelet Volume, Platelet Distribution Width, Plateletcrit, Platelet Large Cell Count and Platelet Large Cell Ratio.

WBC Parameters:

WBC count was considerably lower in those with complications (8366.67 ± 4097.69) versus those in the control group (12504.17 ± 5795.16/µL) with a near significant p-value of 0.056. The Neutrophil percentage was relatively lower in the complication group (67.50 ± 14.50%) in comparison to the control group 74.30 ± 9.69% with p = 0.191. Absolute neutrophil count also showed a comparable trend of 6058.33 ± 4307.17 in complication group against 8461.67 ± 4104.64 in controls, (p = 0.176).The absolute lymphocyte count was lower in the patients with complications (1722.50 ± 717.50) than controls (2029.17 ± 946.45), p = 0.381. However, Lymphocyte percentage was higher in the complication group (23.67 ± 11.89%) when compared to the controls (17.65 ± 8.31%), p = 0.165 (Table 1, Figure 7).

RBC Parameters:

Hemoglobin levels were significantly higher in the group with complications (11.94 ± 1.82 g/dL) in comparison to the control group (9.98 ± 2.19 g/dL), with p value of 0.026., which is statistically significant. HCT was also significantly elevated in the complication group (35.76 ± 5.54%) versus the control group (29.63 ± 7.31%), with p value of 0.030 which is significant. RDW-CV showed slightly lower values in the complication group (14.03 ± 1.79%) compared to control group(14.81 ± 2.70%), but the difference was not significant (p = 0.410) (Table 2, Figure 8).

Platelet Parameters:

Platelet count was marginally lower in the group with complications (273.75 ± 103.68 ×10⁹/L) compared to the control group (305.17 ± 121.60 ×10⁹/L), with p = 0.503. MPV was slightly lower in the group with complications (9.64 ± 0.78 fL) in comparison to the control group (10.05 ± 1.81 fL), with p = 0.480. PDW had relatively unchanged values across groups with 16.04 ± 0.33 in the complication group vs. 16.23 ± 0.29 in controls, p = 0.150. PCT was marginally higher in the complication group (0.262 ± 0.096%) compared to controls (0.248 ± 0.071%), with p = 0.702. P-LCC was slightly higher in the complication group (63416.67 ± 22999.84) than in the control group (60416.67 ± 14425.09), p = 0.706. PLCR values were nearly identical between groups: 23.93 ± 5.18 (complications) vs. 24.13 ± 4.46 (controls), p = 0.920 (Table 3, Figure 9).

Statistically significant differences were observed in hemoglobin and hematocrit, which were higher in the complication group (Table 2). WBC count approached significance and may reflect inflammatory shifts (Table 1). Although platelet

indices did not reach statistical significance, consistent patterns such as slightly reduced MPV and platelet count in the complication group warrant further investigation in larger populations (Table 3).

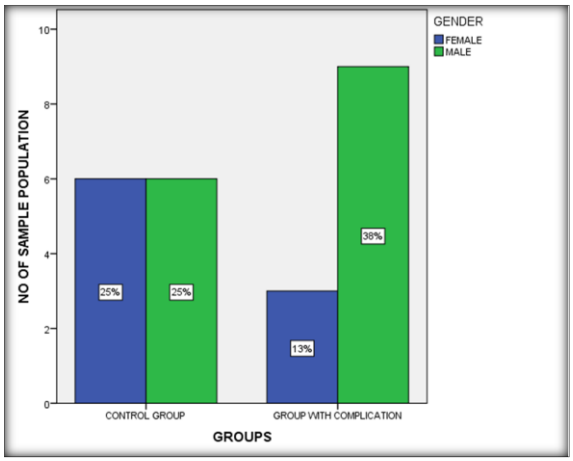

Figure 1: Population distribution in terms of gender

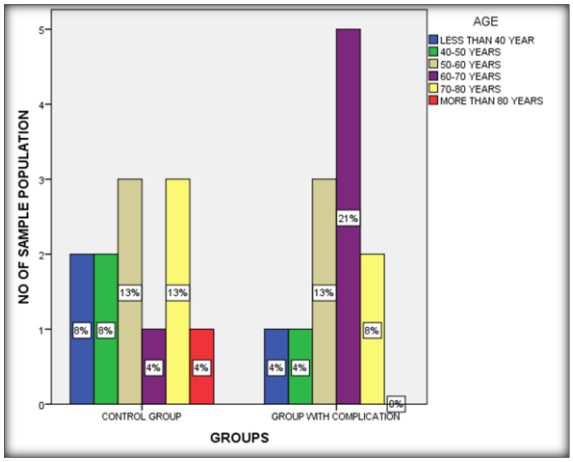

Figure 2: Population distribution in terms of age

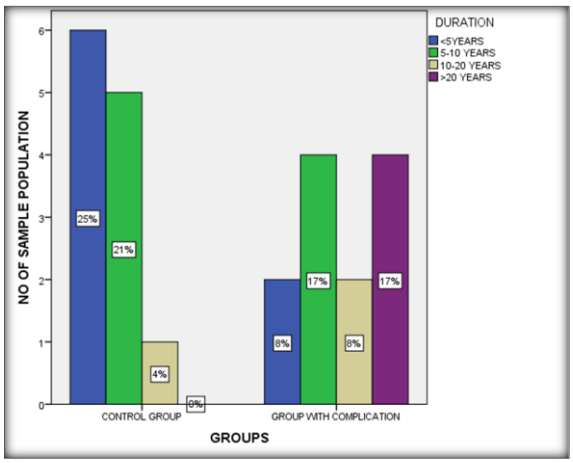

Figure 3: Population distribution in terms of duration of diabetes

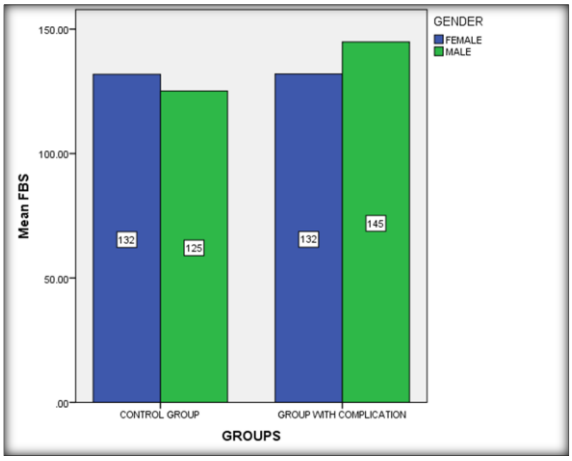

Figure 4: Comparison of mean FBS among control group and group with complications in terms of gender

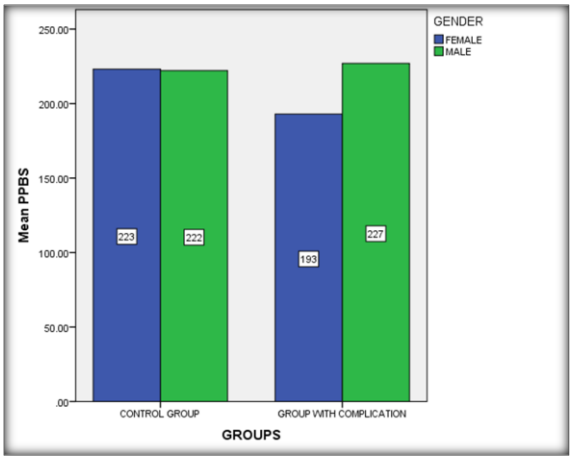

Figure 5: Comparison of mean PPBS among control group and group with complications in terms of gender

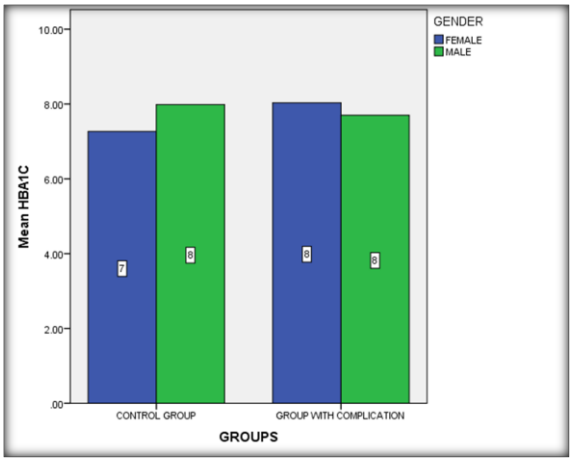

Figure 6: Comparison of mean HbA1C among control group and group with complications in terms of gender

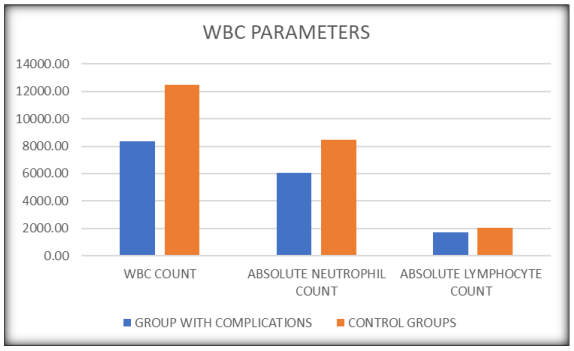

Figure 7: Comparison of WBC parameters among control group and group with complications

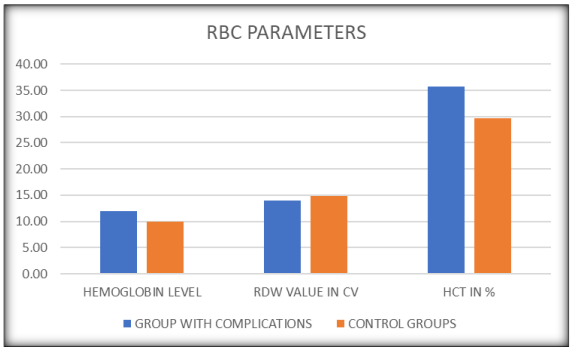

Figure 8: Comparison of RBC parameters among control group and group with complications

Table 1: Comparison of WBC parameters among control group and group with complications

	Groups	N	Mean	Std. Deviation	Std. Error Mean	P. Value
WBC Count	CONTROL GROUP	12	12504.1667	5795.15600	1672.91173	0.056
	GROUP WITH COMPLICATIONS	12	8366.6667	4097.68967	1182.90112	
Neutrophil %	CONTROL GROUP	12	74.3000	9.69170	2.79775	0.191
	GROUP WITH COMPLICATIONS	12	67.5000	14.50078	4.18602	
Absolute Neutrophil Count	CONTROL GROUP	12	8461.6667	4104.63559	1184.90623	0.176
	GROUP WITH COMPLICATIONS	12	6058.3333	4307.17337	1243.37385	
Lymphocyte %	CONTROL GROUP	12	17.6500	8.31171	2.39938	0.165
	GROUP WITH COMPLICATIONS	12	23.6667	11.88837	3.43188	
Absolute Lymphocyte Count	CONTROL GROUP	12	2029.1667	2029.1667	946.45231	0.381
	GROUP WITH COMPLICATIONS	12	1722.5000	1722.5000	717.49723	

Table 2: Comparison of RBC parameters among control group and group with complications

	GROUPS	N	Mean	Std. Deviation	Std. Error Mean	P. Value
Hemoglobin	CONTROL GROUP	12	9.9833	2.18542	0.63088	0.027
	GROUP WITH COMPLICATIONS	12	11.947	1.82281	0.52620	
RDW	CONTROL GROUP	12	14.8083	2.69561	0.77815	0.410
	GROUP WITH COMPLICATIONS	12	14.0250	1.78587	0.51554	
HCT	CONTROL GROUP	12	29.6333	7.30670	2.10926	0.031
	GROUP WITH COMPLICATIONS	12	35.7583	5.53788	1.59865	

Table 3: Comparison of platelet parameters among control group and group with complications

Parameters	GROUPS	N	Mean	Std. Deviation	Std. Error Mean	P. Value
PLATELET COUNT	CONTROL GROUP	12	305.1667	121.59608	35.10176	0.503
	GROUP WITH COMPLICATIONS	12	273.7500	103.68495	29.93127	
MPV	CONTROL GROUP	12	10.0500	1.80630	0.52143	0.480
	GROUP WITH COMPLICATIONS	12	9.6417	0.77748	0.22444	
PDW	CONTROL GROUP	12	16.2333	0.29336	0.8469	0.150
	GROUP WITH COMPLICATIONS	12	16.0417	0.33428	0.9650	
PCT	CONTROL GROUP	12	0.2483	0.7069	0.2041	0.702
	GROUP WITH COMPLICATIONS	12	0.2617	0.09609	0.2774	
P-LCC	CONTROL GROUP	12	60416.6667	14425.09388	4164.16592	0.706
	GROUP WITH COMPLICATIONS	12	63416.6667	22999.83531	6639.48055	
P-LCR	CONTROL GROUP	12	24.1250	4.45954	1.28736	0.920
	GROUP WITH COMPLICATIONS	12	23.9250	5.17882	1.49500	

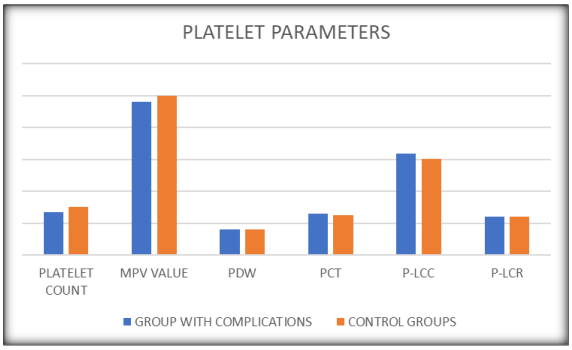

Figure 9: Comparison of platelet parameters among control group and group with complications

DISCUSSION

This pilot study aimed to explore differences in hematological parameters between patients with type 2 diabetes mellitus who had complications and those who did not. While most parameters did not show statistically significant differences, likely due to the small sample size, the observed trends still provide insight into how hematological profiles might shift as complications develop.

White Blood Cell Parameters

Leukocyte count is often considered a practical marker of systemic inflammation. The chronic hyperglycemic environment in diabetes tends to activate inflammatory pathways both systemically and locally. Neutrophils and lymphocytes, which play a key role in innate and adaptive immunity, have been shown to contribute to the development of diabetic microvascular complications. Some studies have observed that patients with complications generally have higher WBC counts, although these often remain within normal laboratory ranges. Additionally, research has highlighted higher neutrophil counts in patients with diabetic nephropathy (DN) and diabetic retinopathy (DR), suggesting a possible link between neutrophil activation and these complications.[20,21]

In our dataset, we noted that the total WBC count and absolute neutrophil count were actually higher in the control group than in the complications group, though these differences were not significant ($p = 0.056$ and $p = 0.176$, respectively). It is possible that higher WBC counts in patients without complications reflect subclinical inflammation at earlier disease stages, whereas lower counts in complicated cases might reflect immune exhaustion or dysregulation that can occur with long-standing disease. Interestingly, lymphocyte percentages and absolute lymphocyte counts were higher in the complications group, again without statistical significance (Table 1). This could represent a compensatory immune response or a shift in immune cell balance associated with chronic complications.

Red Blood Cell Parameters

Previous research has shown that lower hemoglobin levels are common in diabetes and often linked to a higher prevalence of DR and DN. For example, Redondo-Bermejo et al,[22] found that red blood cell survival decreases by about 13% in hyperglycemia. Qiao et al,[23] reported an odds ratio of 2.0 for DR in patients whose hemoglobin was below 120 g/L, and Davis et al,[24] found that lower hematocrit was associated with an increased risk of high-risk proliferative DR over five years (OR 1.52). Several other studies echo this association between reduced hemoglobin and DR in type 2 diabete.[23,25] One hypothesis suggests that low hemoglobin reduces shear stress in small vessels, which in turn decreases nitric oxide synthesis and affects vessel tone and angiogenesis. Chronic anemia in diabetes is also often related to diabetic kidney disease.[26,27]

Interestingly, our findings contrast with these earlier reports. We observed significantly higher hemoglobin ($p = 0.026$) and hematocrit ($p = 0.030$) levels in patients with complications. One possible explanation is a compensatory increase in red cell production driven by tissue hypoxia early in the course of vascular disease. Alternatively, factors like dehydration, relative polycythemia, or subtle plasma volume shifts related to nephropathy or autonomic dysfunction could also play a role. As for red cell distribution width (RDW), although our data showed higher RDW in the control group, this difference was not statistically significant ($p = 0.410$) (Table 2). Since RDW has been linked to inflammation and cardiovascular risk, it remains an interesting parameter for future larger studies.

Platelet Parameters

Diabetes is recognized as a prothrombotic condition, largely because of increased platelet reactivity, accelerated turnover, and morphological changes.[19,16] Larger and younger platelets, reflected by higher mean platelet volume (MPV) and platelet distribution width (PDW), are thought to play a role in vascular complications.[16,17] Hyperglycemia and oxidative stress can worsen this activation, leading to complications like DR, DN, and neuropathy.

Several studies back up the clinical usefulness of platelet indices. Taderegew et al. found significantly higher MPV (13.57 ± 2.17 fL vs. 11.76 ± 1.93 fL) and PDW (16.57 ± 2.49 fL vs. 14.97 ± 2.41 fL) in patients with complications, with these indices also showing independent associations in multivariate analysis (AOR=1.68 and 1.37) (28). Walinjkar et al. reported similar increases in MPV, PDW, and platelet-large cell ratio (P-LCR) among diabetics with complications,[17] and Jindal et al. found higher PDW in complicated cases (P=0.006).[29] (Table 3).

In our pilot study, however, we did not observe significant differences in platelet count, MPV, PDW, PCT, PLCC, or PLCR between the two groups. MPV and PDW are often elevated in inflammatory and pro-thrombotic states linked to diabetic complications. The lack of significant difference might be due to our limited sample size, but the observation of slightly lower MPV and PDW in the complication group could hint at chronic low-grade activation or platelet exhaustion. Prior studies have suggested that higher MPV and PDW may predict complications like

retinopathy and nephropathy by reflecting increased platelet turnover and endothelial interaction. Although our data do not prove this relationship, the trend we observed supports this hypothesis and highlights the need for further research.

While our pilot study was small and underpowered, it uncovered subtle yet potentially meaningful differences in hematological parameters between diabetic patients with and without complications. Higher hemoglobin and hematocrit levels in complicated cases, along with trends in WBC and platelet indices, suggest possible shifts in the inflammatory and hematopoietic profile as diabetes progresses.

Future studies should aim for larger sample sizes and include stratification by type of complication, duration of diabetes, and glycemic control (e.g., HbA1c levels). Longitudinal designs would also help clarify whether these hematological markers can truly predict the onset or progression of complications. Such research could eventually lead to practical, cost-effective tools to identify high-risk patients earlier in the disease course.

CONCLUSION

This pilot study set out to explore differences in hematological parameters between type 2 diabetic patients with and without complications. Although most differences were not statistically significant, likely due to the small sample size, we did observe patterns that might point to real biological trends. Higher hemoglobin and hematocrit levels in patients with complications could reflect adaptive or pathological changes related to disease progression. Similarly, subtle variations in white blood cell and platelet indices hint at shifts in immune activity and thrombosis risk as complications emerge. Given the limited number of participants, these findings should be interpreted with caution. Still, they suggest that everyday hematological markers, which are inexpensive and widely available, could potentially help identify diabetic patients at higher risk for complications. Future research involving larger, well-designed cohorts will be essential to confirm these trends and determine whether such parameters can be used reliably for early detection or risk prediction in clinical practice.

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