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Association of Red Cell Distribution Width with Glycemic Control and Cardiometabolic Risk Factors in Type 2 Diabetes Mellitus

Dr. Girish Thawani*, Dr. Vivek Katiyar, Dr. Priyanka Singh, Dr. Vishal Parmar, Dr. Tauseef Khan

Junior Resident[1], Department of General Medicine, Integral Institute of Medical Sciences and Research, Lucknow, Uttar Pradesh, India.
Professor[2], Department of General Medicine, Integral Institute of Medical Sciences and Research, Lucknow, Uttar Pradesh, India.
Professor and HOD[3], Department of Pathology, Integral Institute of Medical Sciences and Research, Lucknow, Uttar Pradesh, India.
Professor and HOD[4], Department of General Medicine, Integral Institute of Medical Sciences and Research, Lucknow, Uttar Pradesh, India.
Assistant Professor[5], Dept of general medicine, Integral Institute of Medical Sciences and Research, Lucknow, Uttar Pradesh, India.

Corresponding Author: Dr. Girish Thawani*,
Email ID: girishthawani11@gmail.com

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder associated with persistent hyperglycemia and a wide spectrum of microvascular and macrovascular complications. Identifying simple, cost-effective biomarkers that can predict poor glycemic control is crucial for early risk stratification and improved disease management. Red cell distribution width (RDW), a routinely reported hematological parameter, has recently gained attention as a potential marker of inflammation and metabolic dysregulation.

Objectives: To evaluate the association of red cell distribution width with glycemic control and to assess its role as an independent predictor of poor glycemic control in patients with type 2 diabetes mellitus.

Materials and Methods: This cross-sectional study was conducted over an 18-month period at a tertiary care center in Lucknow. A total of 120 patients with T2DM were enrolled and divided into two groups: cases (poor glycemic control) and controls (better glycemic control), with 60 participants in each group. Clinical evaluation, anthropometric measurements, and laboratory investigations including HbA1c, fasting and post-prandial blood glucose, lipid profile, hemoglobin, and RDW were performed. Statistical analysis included correlation analysis, ROC curve analysis, and multivariate logistic regression to identify independent predictors of poor glycemic control.

Results: Patients with poor glycemic control had significantly higher RDW levels compared to controls (15.69 ± 0.84 vs 14.29 ± 1.49; $p < 0.0001$). RDW showed a significant positive correlation with HbA1c, fasting blood sugar, and post-prandial blood sugar ($p < 0.0001$). ROC curve analysis demonstrated good diagnostic accuracy of RDW for predicting poor glycemic control (AUC = 0.76). On multivariate analysis, RDW emerged as an independent predictor of poor glycemic control (OR = 1.28; 95% CI: 1.12–1.46; $p < 0.0001$), even after adjusting for confounding factors.

Conclusion: Red cell distribution width is significantly associated with poor glycemic control and serves as an independent predictor in patients with type 2 diabetes mellitus. Given its low cost and routine availability, RDW may be used as an adjunctive biomarker for identifying high-risk diabetic patients.

KEYWORDS: Type 2 diabetes mellitus, Red cell distribution width, Glycemic control, HbA1c, Biomarker.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia due to insulin resistance and relative insulin deficiency. It represents a major global health burden, with rapidly increasing prevalence, particularly in low- and middle-income countries such as India [1]. According to the International Diabetes Federation, India is among the countries with the highest number of individuals living with diabetes, and this number is projected to rise substantially in the coming decades [2]. Poor glycemic control in T2DM is strongly associated with the development of

microvascular complications such as diabetic retinopathy, nephropathy, and neuropathy, as well as macrovascular complications including coronary artery disease, stroke, and peripheral vascular disease [3,4].

Glycated hemoglobin (HbA1c) is widely accepted as the gold standard marker for assessing long-term glycemic control and for predicting the risk of diabetic complications [5]. However, HbA1c measurement has certain limitations, as it can be influenced by conditions affecting red blood cell turnover, hemoglobinopathies, anemia, chronic kidney disease, and recent blood transfusions [6]. Furthermore, in resource-limited settings, access to HbA1c testing may be restricted due to cost and availability. These limitations have prompted interest in identifying alternative or adjunctive biomarkers that are inexpensive, easily accessible, and capable of reflecting metabolic and inflammatory changes associated with diabetes [7].

Red cell distribution width (RDW) is a routinely reported hematological parameter that reflects the variability in the size of circulating erythrocytes (anisocytosis). Traditionally, RDW has been used in the differential diagnosis of various types of anemia [8]. In recent years, however, RDW has gained considerable attention as a novel prognostic marker in a variety of non-hematological conditions, including cardiovascular diseases, chronic kidney disease, metabolic syndrome, and inflammatory disorders [9–11]. Elevated RDW has been shown to be associated with increased morbidity and mortality in patients with heart failure and coronary artery disease, independent of traditional risk factors [12].

The association between RDW and diabetes-related outcomes is believed to be mediated through several pathophysiological mechanisms. Chronic hyperglycemia induces oxidative stress and low-grade systemic inflammation, which adversely affect erythropoiesis and reduce red blood cell lifespan, leading to increased heterogeneity in red blood cell size and elevated RDW values [13,14]. Inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha may further suppress erythropoietin activity and impair iron metabolism, contributing to anisocytosis [15]. Additionally, insulin resistance and dyslipidemia, which commonly accompany poor glycemic control, have been linked to altered red blood cell membrane structure and function [16].
Several observational studies have reported a significant association between elevated RDW and poor glycemic control, as reflected by higher HbA1c levels in patients with T2DM [17,18]. Higher RDW values have also been associated with increased prevalence of hypertension, dyslipidemia, obesity, and metabolic syndrome in diabetic individuals, suggesting that RDW may serve as an integrated marker of cardiometabolic risk [19,20]. Moreover, emerging evidence suggests that RDW may predict adverse cardiovascular outcomes and all-cause mortality in patients with diabetes [21].

Despite growing evidence supporting the clinical relevance of RDW, data regarding its role as an independent predictor of poor glycemic control in Indian patients with type 2 diabetes mellitus remain limited. Given the simplicity, low cost, and widespread availability of RDW as part of routine complete blood count testing, establishing its utility could have significant implications for risk stratification and early identification of poorly controlled diabetic patients in routine clinical practice.

Therefore, the present study was undertaken to evaluate the association between red cell distribution width and glycemic parameters in patients with type 2 diabetes mellitus. The study further aimed to assess the diagnostic performance of RDW in predicting poor glycemic control and to determine whether RDW remains an independent predictor after adjusting for relevant demographic, clinical, and metabolic confounders.

MATERIAL AND METHODS

This was a hospital-based cross-sectional analytical study conducted over a period of 18 months in the Department of General Medicine, in collaboration with the Department of Biochemistry, at Integral Institute of Medical Sciences and Research (IIMS&R), Lucknow.

Study Population

The study included adult patients with a confirmed diagnosis of Type 2 Diabetes Mellitus (T2DM) attending the outpatient and inpatient services of the Department of Medicine. A total of 120 participants were enrolled and divided into two groups:

- Cases (n = 60): Patients with poor glycemic control
- Controls (n = 60): Patients with comparatively better glycemic control

Grouping was based on HbA1c levels, as per standard clinical guidelines.

Sampling Technique

Participants were selected using a non-probability random sampling technique after fulfilling the eligibility criteria.

Inclusion Criteria

Participants fulfilling all of the following criteria were included in the study:

1. Adults aged 18 to 75 years.
2. Confirmed diagnosis of Type 2 Diabetes Mellitus based on standard diagnostic criteria.
3. Availability of recent HbA1c estimation.
4. Stable antidiabetic treatment regimen for at least three months prior to enrollment.
5. Hemoglobin level >11.0 g/dL.
6. Willingness to participate in the study and provision of written informed consent.

Exclusion Criteria
Participants meeting any of the following criteria were excluded:
1. Patients with Type 1 diabetes mellitus or secondary forms of diabetes.
2. Presence of chronic kidney disease.
3. Known hematological disorders affecting red blood cell indices, such as thalassemia or hemophilia.
4. Pregnant or breastfeeding women.
5. History of recent major cardiovascular events (myocardial infarction, stroke) within the past six months.
6. Presence of severe systemic illnesses or comorbid conditions likely to affect study outcomes.
7. Recent blood transfusion within the last three months.

Data Collection and Clinical Evaluation
After obtaining informed consent, all participants underwent a detailed clinical evaluation including demographic details, duration of diabetes, history of comorbidities, medication history, and lifestyle factors. Anthropometric measurements such as body mass index (BMI) and waist circumference were recorded using standard techniques. Blood pressure was measured in a seated position after adequate rest.

Laboratory Investigations
Venous blood samples were collected after an overnight fast. The following investigations were performed:
- Fasting blood glucose (FBS)
- Post-prandial blood glucose (PPBS)
- Glycated hemoglobin (HbA1c)
- Complete blood count including red cell distribution width (RDW) and hemoglobin
- Fasting lipid profile

All laboratory analyses were performed in the central laboratory following standardized protocols.

RESULTS

A total of 120 patients with type 2 diabetes mellitus were included in the study, comprising 60 cases with poor glycemic control and 60 controls with comparatively better glycemic control. Both groups were comparable in terms of age distribution, with the majority of participants belonging to the 51–60 years age group, followed by the 61–70 years group. The overall age distribution between cases and controls did not show a statistically significant difference ($\chi^2 = 1.02$, $p = 0.960$), indicating appropriate matching of study groups.

The mean age of the cases was 55.98 ± 12.08 years, which was comparable to that of the controls (56.47 ± 10.83 years; $p = 0.820$). However, the mean duration of diabetes was significantly longer among cases (9.15 ± 3.73 years) compared to controls (5.48 ± 2.37 years), and this difference was highly statistically significant ($p < 0.0001$). Anthropometric assessment revealed that cases had significantly higher body mass index (28.59 ± 3.32 kg/m² vs 25.59 ± 3.28 kg/m²; $p < 0.0001$) and waist circumference (96.57 ± 7.41 cm vs 88.47 ± 6.51 cm; $p < 0.0001$), indicating greater central obesity among patients with poor glycemic control.

Gender distribution showed no significant difference between the two groups, with males constituting 51.7% of cases and 63.3% of controls ($p = 0.196$). The prevalence of hypertension was significantly higher among cases (73.3%) compared to controls (45.0%) ($p = 0.002$). Similarly, hyperlipidemia was more commonly observed in cases (53.3%) than in controls (31.7%), and this difference was statistically significant ($p = 0.017$). Coronary artery disease was present in 30.0% of cases as compared to 10.0% of controls, showing a significant association with poor glycemic control ($p = 0.006$). Chronic kidney disease was noted in 11.7% of cases, while none of the controls had CKD, and this difference was statistically significant using Fisher's exact test ($p = 0.013$). Metabolic syndrome was significantly more prevalent among cases (68.3%) than controls (43.3%) ($p = 0.006$).

Glycemic parameters were markedly deranged in cases compared to controls. The mean HbA1c level was significantly higher in cases (8.99 ± 0.85%) compared to controls (6.30 ± 0.45%) ($p < 0.0001$). Similarly, fasting blood sugar (183.92 ± 34.26 mg/dL vs 113.26 ± 20.82 mg/dL) and post-prandial blood sugar levels (258.21 ± 46.00 mg/dL vs 166.55 ± 27.80 mg/dL) were significantly higher among cases, with both comparisons showing highly significant p-values (<0.0001).

Lipid profile analysis demonstrated a significantly more atherogenic pattern in cases. Total cholesterol, triglycerides, and low-density lipoprotein (LDL) cholesterol levels were significantly higher among cases, while high-density lipoprotein (HDL) cholesterol levels were significantly lower compared to controls ($p < 0.0001$ for all parameters). These findings indicate a strong association between poor glycemic control and dyslipidemia.

Hematological analysis revealed that mean hemoglobin levels were slightly but significantly lower in cases (13.05 ± 1.06 g/dL) compared to controls (13.54 ± 0.92 g/dL) ($p = 0.007$). In contrast, mean red cell distribution width (RDW) was significantly higher in cases (15.69 ± 0.84%) compared to controls (14.29 ± 1.49%), with a highly significant difference ($p < 0.0001$).

Correlation analysis showed a moderate positive correlation between RDW and HbA1c ($r = 0.417$, $p < 0.0001$), indicating that higher RDW values were associated with poorer long-term glycemic control. RDW also demonstrated significant positive

correlations with fasting blood sugar ($r = 0.320$, $p < 0.0001$) and post-prandial blood sugar ($r = 0.394$, $p < 0.0001$). Additionally, HbA1c showed a positive correlation with triglyceride levels and a negative correlation with HDL cholesterol, both of which were statistically significant ($p < 0.0001$).

Receiver operating characteristic (ROC) curve analysis demonstrated that RDW had good diagnostic accuracy for predicting poor glycemic control, with an area under the curve of 0.76 (95% CI: 0.68–0.83; $p < 0.0001$). An optimal RDW cut-off value of 14.8% yielded a sensitivity of 71% and specificity of 66%, indicating a reasonable balance between true positive and true negative rates.

On multivariate binary logistic regression analysis, RDW emerged as a strong independent predictor of poor glycemic control (OR = 1.28; 95% CI: 1.12–1.46; $p < 0.0001$). Other significant predictors included age, duration of diabetes, body mass index, waist circumference, triglyceride levels, low HDL cholesterol, lower hemoglobin levels, and the presence of metabolic syndrome. Male gender and hypertension did not show statistically significant independent associations. Even after adjustment for multiple confounding variables, RDW remained independently associated with poor glycemic control.

TABLE 1: Distribution of Study Participants

Group	Number (n)	Percentage (%)
Cases	60	50.0
Controls	60	50.0
Total	**120**	**100**

A total of 120 participants were included in the study, of which 60 subjects (50.0%) were categorized as cases and 60 subjects (50.0%) as controls.

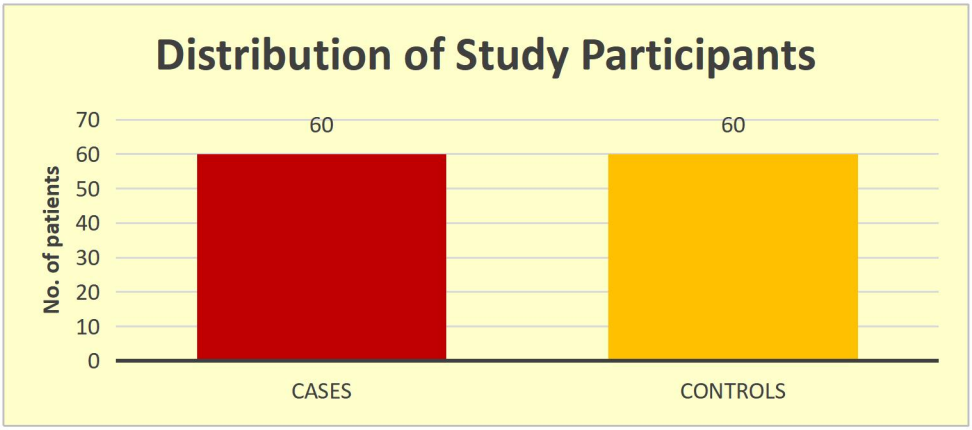

Figure: 1 Graphical representations of Distribution of Study participants

TABLE 2: Age Distribution of Study Participants

Age Group (years)	Cases n (%)	Controls n (%)	χ^2 value	p-value
18–30	3 (5.0)	1 (1.7)	**1.02**	**0.960**
31–40	4 (6.7)	3 (5.0)		
41–50	12 (20.0)	13 (21.7)		
51–60	22 (36.7)	20 (33.3)		
61–70	14 (23.3)	17 (28.3)		
>70	5 (8.3)	6 (10.0)		

The majority of participants in both groups were concentrated in the 51–60 years age group, comprising 22 cases (36.7%) and 20 controls (33.3%), followed by the 61–70 years age group with 14 cases (23.3%) and 17 controls (28.3%). Participants aged 41–50 years accounted for 12 cases (20.0%) and 13 controls (21.7%), while younger age groups 18–30 years and 31–40 years constituted a smaller proportion of the study population. The oldest age group (>70 years) included 5 cases (8.3%) and 6 controls (10.0%).

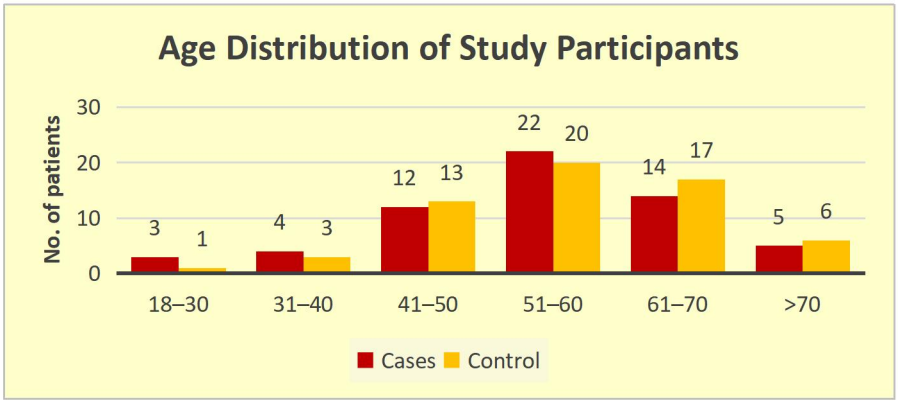

Figure: 2 Graphical representations of age distribution of study participants

TABLE 3: Comparison of Demographic &Anthropometric Parameters

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	t value	p-value
Age (years)	55.98 ± 12.08	56.47 ± 10.83	−0.23	0.820
Duration of diabetes (years)	9.15 ± 3.73	5.48 ± 2.37	6.44	**<0.0001***
BMI (kg/m²)	28.59 ± 3.32	25.59 ± 3.28	5.01	**<0.0001***
Waist circumference (cm)	96.57 ± 7.41	88.47 ± 6.51	6.35	**<0.0001***

The waist circumference was also markedly higher in cases (96.57 ± 7.41 cm) compared to controls (88.47 ± 6.51 cm), and this difference was statistically highly significant ($t = 6.35$, $p < 0.0001$), indicating greater adiposity among cases.

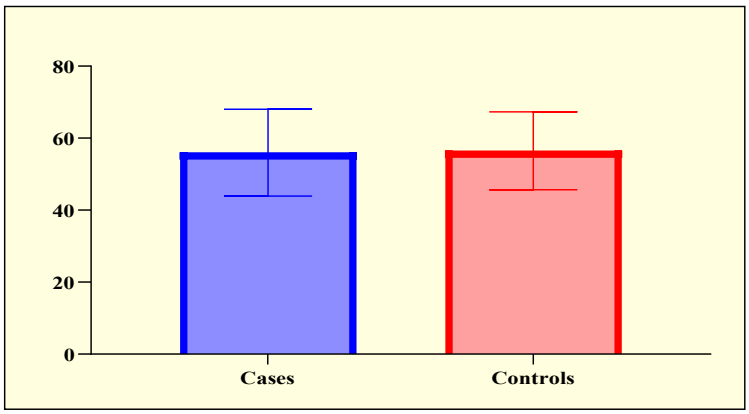

Figure: 3 Graphical representations of Comparison of Demographic & Anthropometric Parameters.

TABLE 4: Gender Distribution

Gender	Cases n (%)	Controls n (%)	χ^2 value	p-value
Male	31 (51.7)	38 (63.3)	**1.67**	**0.196**
Female	29 (48.3)	22 (36.7)		

The gender distribution of the study participants. Among the cases, 31 participants (51.7%) were males and 29 participants (48.3%) were females, whereas in the control group 38 participants (63.3%) were males and 22 participants (36.7%) were females.

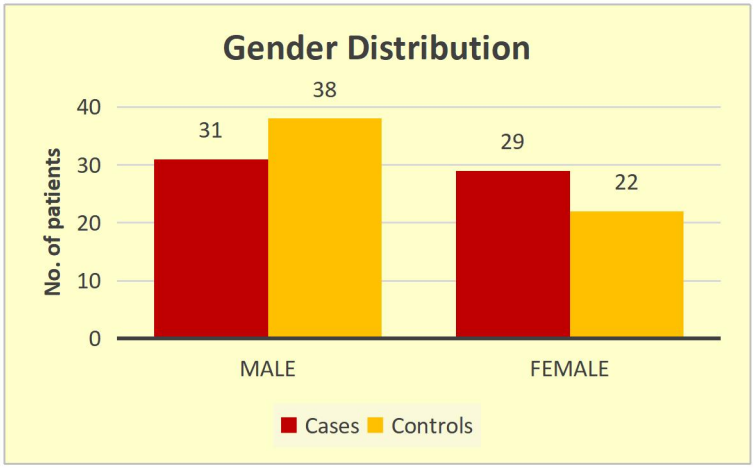

Figure: 4 Graphical representations Gender Distribution

TABLE 5: Distribution of Hypertension

Hypertension	Cases n (%)	Controls n (%)	χ^2 value	p-value
Yes	44 (73.3)	27 (45.0)		
No	16 (26.7)	33 (55.0)	**9.84**	**0.002***

Hypertension was present in 44 cases (73.3%) compared to 27 controls (45.0%), while 16 cases (26.7%) and 33 controls (55.0%) were normotensive. The prevalence of hypertension was thus markedly higher among cases than controls. This difference was statistically significant, with a chi-square value of 9.84 and a p-value of 0.002, indicating a strong association between case status and the presence of hypertension.

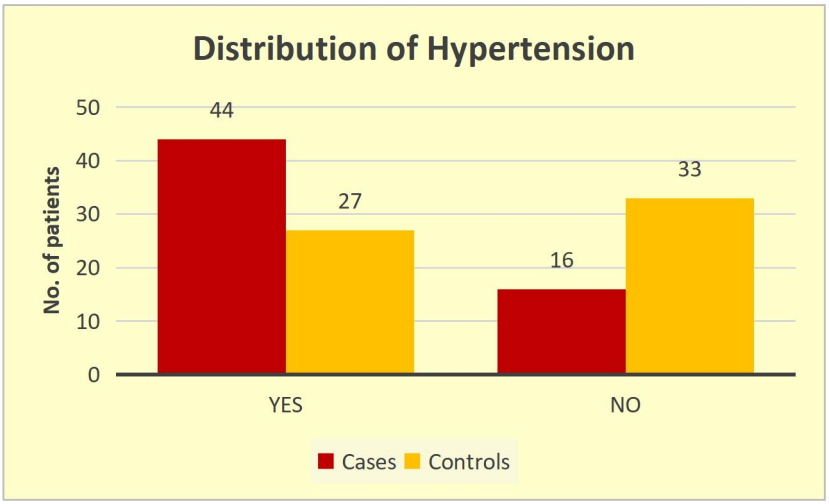

Figure: 5 Graphical representations of Distribution of Hypertension.

TABLE 6: Distribution of Hyperlipidemia

Hyperlipidemia	Cases n (%)	Controls n (%)	χ^2 value	p-value
Yes	32 (53.3)	19 (31.7)		
No	28 (46.7)	41 (68.3)	**5.69**	**0.017***

Hyperlipidemia was observed in 32 cases (53.3%) compared to 19 controls (31.7%), whereas 28 cases (46.7%) and 41 controls (68.3%) did not have hyperlipidemia. The proportion of participants with hyperlipidemia was therefore higher among cases than controls. This difference was statistically significant, with a chi-square value of 5.69 and a p-value of 0.017, suggesting a significant association between case status and the presence of hyperlipidemia.

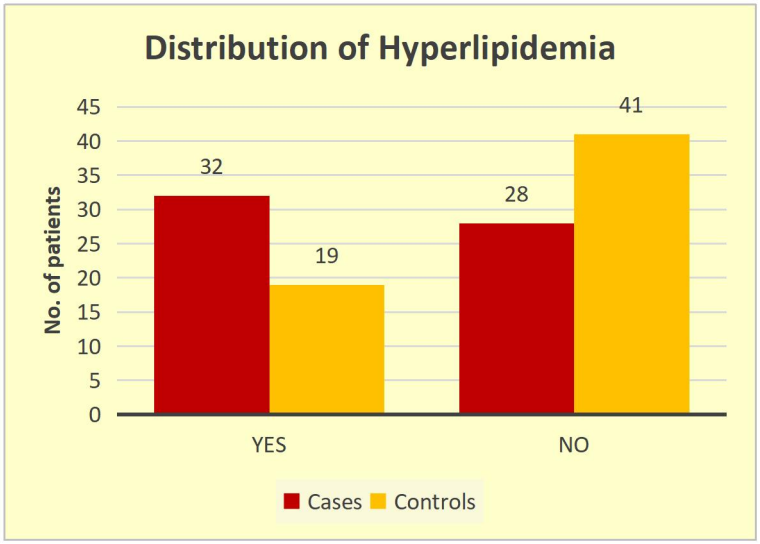

Figure: 6 Graphical representations of Distribution of Hyperlipidemia

TABLE 7: Distribution of Coronary Artery Disease

CAD	Cases n (%)	Controls n (%)	χ^2 value	p-value
Yes	18 (30.0)	6 (10.0)		
No	42 (70.0)	54 (90.0)	**7.50**	**0.006***

The distribution of coronary artery disease (CAD) among cases and controls. CAD was present in 18 cases (30.0%) compared to 6 controls (10.0%), while 42 cases (70.0%) and 54 controls (90.0%) did not have CAD. The prevalence of CAD was therefore substantially higher among cases than controls. This difference was statistically significant, with a chi-square value of 7.50 and a p-value of 0.006, indicating a significant association between case status and the presence of coronary artery disease.

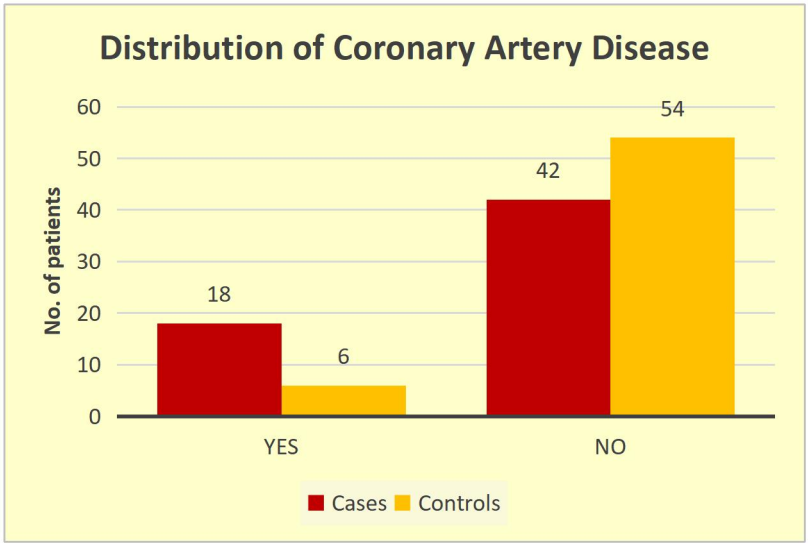

Figure: 7 Graphical representations of Distribution of Coronary Artery Disease

TABLE 8: Distribution of Chronic Kidney Disease

CKD	Cases n (%)	Controls n (%)	Test	p-value
Yes	7 (11.7)	0 (0.0)	Fisher	**0.013***
No	53 (88.3)	60 (100)		

The distribution of chronic kidney disease (CKD) among cases and controls. CKD was present in 7 cases (11.7%), while no control participants (0.0%) had CKD; conversely, 53 cases (88.3%) and all 60 controls (100%) were free from CKD. Due to the

presence of zero values in the control group, Fisher's exact test was applied, which demonstrated a statistically significant difference between the two groups (p = 0.013), indicating a significant association between case status and the presence of chronic kidney disease.

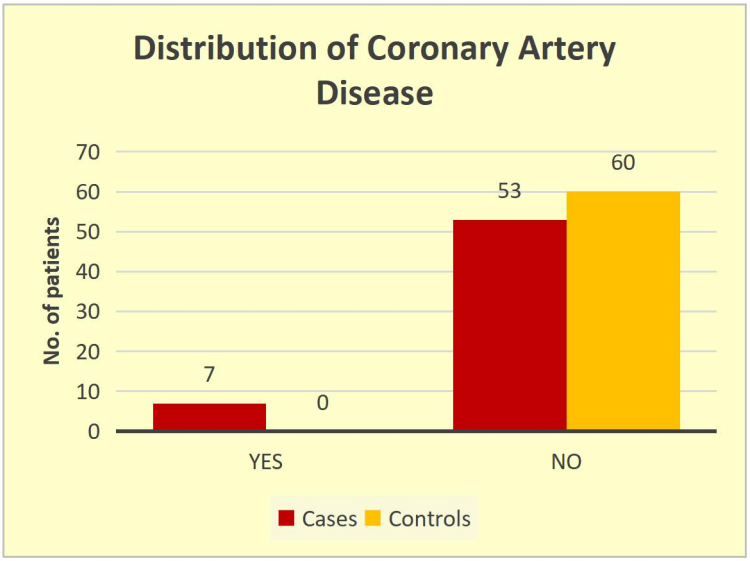

Figure: 8 Graphical representations of Distribution of Chronic Kidney Disease

TABLE 9: Distribution of Metabolic Syndrome

Metabolic Syndrome	Cases n (%)	Controls n (%)	χ^2 value	p-value
Yes	41 (68.3)	26 (43.3)		
No	19 (31.7)	34 (56.7)	**7.62**	**0.006***

The distribution of metabolic syndrome among cases and controls. Metabolic syndrome was present in 41 cases (68.3%) compared to 26 controls (43.3%), while 19 cases (31.7%) and 34 controls (56.7%) did not have metabolic syndrome. The prevalence of metabolic syndrome was notably higher among cases than controls. This difference was statistically significant, with a chi-square value of 7.62 and a p-value of 0.006, indicating a significant association between case status and the presence of metabolic syndrome.

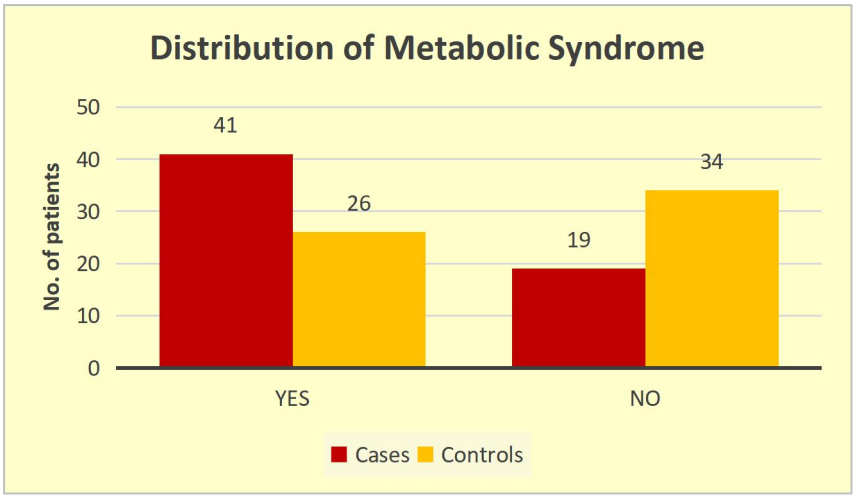

Figure: 9 Graphical representations of Distribution of Metabolic Syndrome

TABLE 10: Comparison of Glycemic Parameters

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	t value	p-value
HbA1c (%)	8.99 ± 0.85	6.30 ± 0.45	21.30	**<0.0001***
FBS (mg/dL)	183.92 ± 34.26	113.26 ± 20.82	13.80	**<0.0001***
PPBS (mg/dL)	258.21 ± 46.00	166.55 ± 27.80	13.20	**<0.0001***

The mean HbA1c level was significantly higher among cases (8.99 ± 0.85%) compared to controls (6.30 ± 0.45%), with a highly significant difference ($t = 21.30$, $p < 0.0001$), indicating poorer long-term glycemic control in cases. Similarly, the mean fasting blood sugar (FBS) was markedly elevated in cases (183.92 ± 34.26 mg/dL) compared to controls (113.26 ± 20.82 mg/dL) ($t = 13.80$, $p < 0.0001$). The mean post-prandial blood sugar (PPBS) levels were also significantly higher among cases (258.21 ± 46.00 mg/dL) than controls (166.55 ± 27.80 mg/dL), with this difference being highly significant ($t = 13.20$, $p < 0.0001$), collectively confirming significantly poorer glycemic control in the case group.

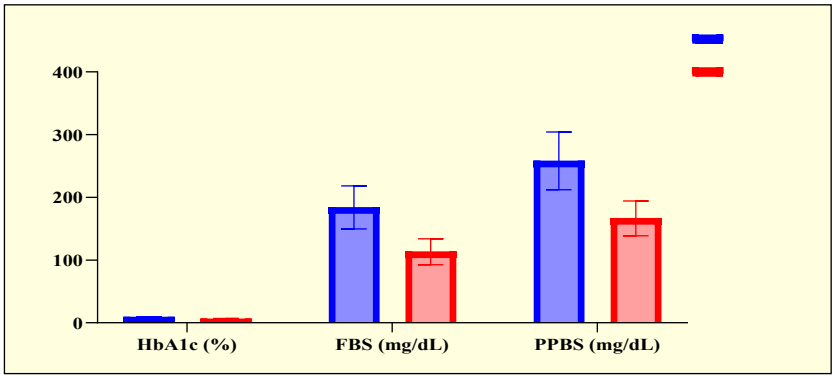

Figure: 10 Graphical representations of Comparison of Glycemic Parameters.

TABLE 11: Comparison of Lipid Profile

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	t value	p-value
Total Cholesterol	222.26 ± 33.18	188.85 ± 19.46	6.60	**<0.0001***
Triglycerides	181.52 ± 32.79	140.78 ± 36.25	6.42	**<0.0001***
HDL	38.46 ± 4.33	43.89 ± 4.44	−6.70	**<0.0001***
LDL	142.89 ± 26.77	120.89 ± 24.53	4.68	**<0.0001***

The mean total cholesterol level was significantly higher in cases (222.26 ± 33.18 mg/dL) compared to controls (188.85 ± 19.46 mg/dL), showing a highly significant difference ($t = 6.60$, $p < 0.0001$). Similarly, mean triglyceride levels were elevated among cases (181.52 ± 32.79 mg/dL) compared to controls (140.78 ± 36.25 mg/dL) ($t = 6.42$, $p < 0.0001$). In contrast, the mean HDL cholesterol level was significantly lower in cases (38.46 ± 4.33 mg/dL) than in controls (43.89 ± 4.44 mg/dL), with a statistically significant difference ($t = -6.70$, $p < 0.0001$). The mean LDL cholesterol was also significantly higher in cases (142.89 ± 26.77 mg/dL) compared to controls (120.89 ± 24.53 mg/dL) ($t = 4.68$, $p < 0.0001$), indicating a more atherogenic lipid profile among cases.

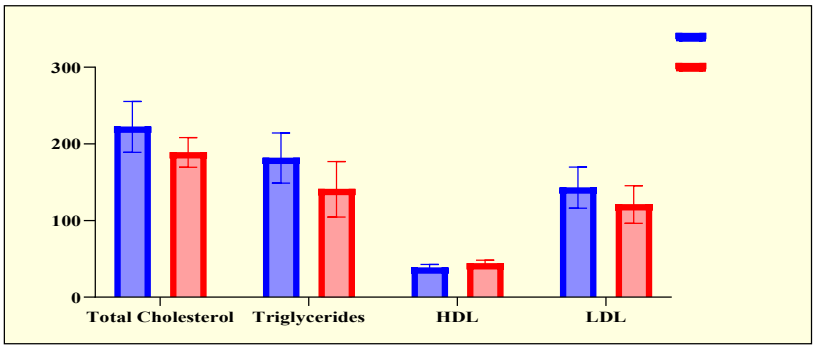

Figure: 11 Graphical representations of Comparison of Lipid Profile

TABLE 12: Comparison of Hematological Parameters

Parameter	Cases (Mean ± SD)	Controls (Mean ± SD)	t value	p-value
Hemoglobin (g/dL)	13.05 ± 1.06	13.54 ± 0.92	−2.74	**0.007***
RDW (%)	15.69 ± 0.84	14.29 ± 1.49	6.17	**<0.0001***

The mean hemoglobin level was slightly but significantly lower in cases (13.05 ± 1.06 g/dL) compared to controls (13.54 ± 0.92 g/dL), with a statistically significant difference ($t = -2.74$, $p = 0.007$). In contrast, the mean red cell distribution width (RDW) was markedly higher among cases (15.69 ± 0.84%) than controls (14.29 ± 1.49%), and this difference was highly statistically significant ($t = 6.17$, $p < 0.0001$), supporting the association of elevated RDW with poor glycemic control in patients with type 2 diabetes mellitus.

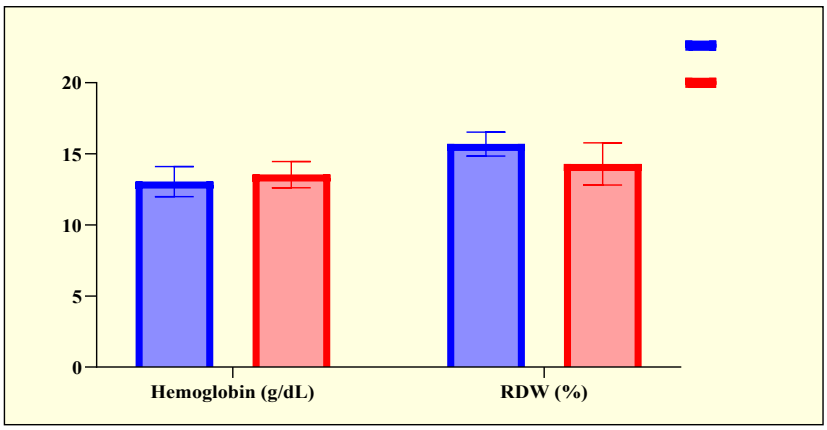

Figure: 12 Graphical representations of Comparison of Hematological Parameters.

TABLE 13: Correlation of RDW with Glycemic Parameters

Parameter	r value	95% CI	p-value
HbA1c (%)	0.417	0.257 – 0.555	**<0.0001***
FBS (mg/dL)	0.320	0.149 – 0.472	**<0.0001***
PPBS (mg/dL)	0.394	0.231 – 0.535	**<0.0001***

The correlation between red cell distribution width (RDW) and glycemic parameters. RDW showed a moderate positive correlation with HbA1c ($r = 0.417$; 95% CI: 0.257–0.555; $p < 0.0001$), indicating that higher RDW values are associated with poorer long-term glycemic control. Similarly, RDW was positively correlated with fasting blood sugar (FBS) ($r = 0.320$; 95% CI: 0.149–0.472; $p < 0.0001$) and post-prandial blood sugar (PPBS) ($r = 0.394$; 95% CI: 0.231–0.535; $p < 0.0001$).

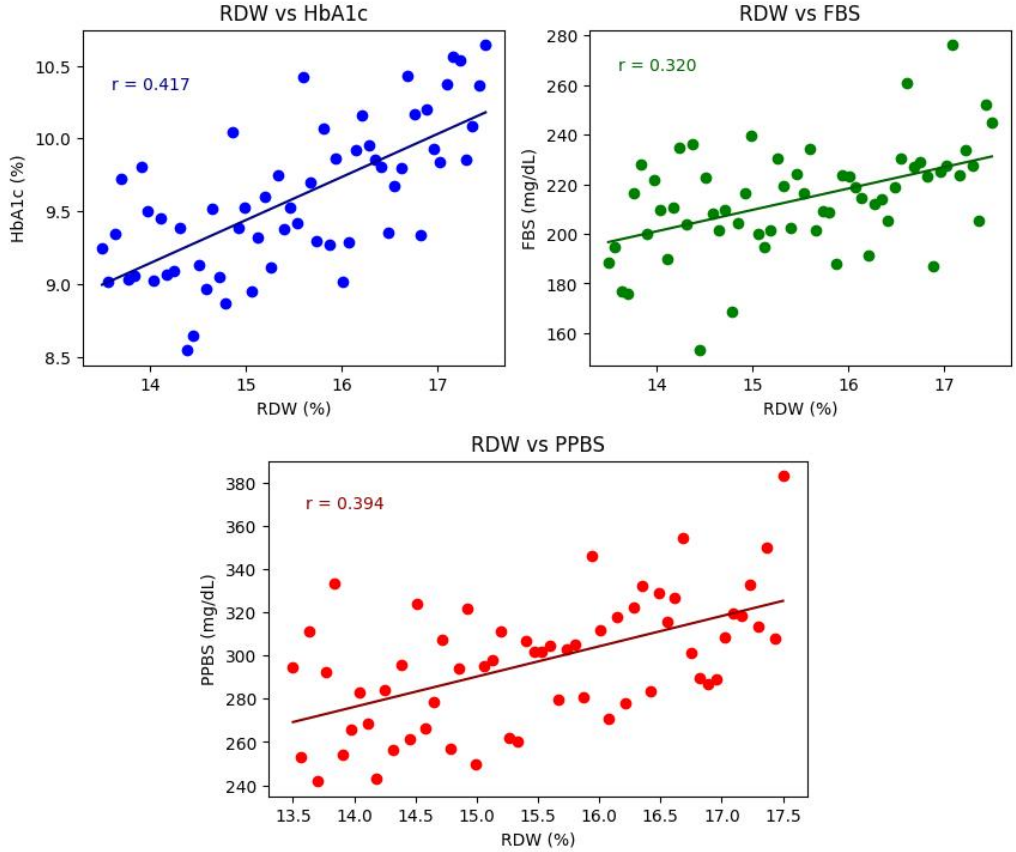

Figure: 13 Scatterplot of the correlation of the RDW with various variables.

TABLE 14: Key Correlations of HbA1c with Selected Variables

Variable	r value	95% CI	p-value
RDW (%)	0.417	0.257 – 0.555	**<0.0001***
Triglycerides	0.468	0.315 – 0.597	**<0.0001***
HDL (mg/dL)	−0.518	−0.638 – −0.373	**<0.0001***

The key correlations of HbA1c with selected metabolic and hematological variables. HbA1c showed a moderate positive correlation with RDW (r = 0.417; 95% CI: 0.257–0.555; p < 0.0001), indicating that higher RDW levels are associated with poorer glycemic control. A stronger positive correlation was observed between HbA1c and triglyceride levels (r = 0.468; 95% CI: 0.315–0.597; p < 0.0001), suggesting worsening dyslipidemia with increasing glycemic levels. In contrast, HbA1c demonstrated a significant negative correlation with HDL cholesterol (r = −0.518; 95% CI: −0.638 to −0.373; p < 0.0001), indicating that higher HbA1c levels are associated with lower protective HDL levels.

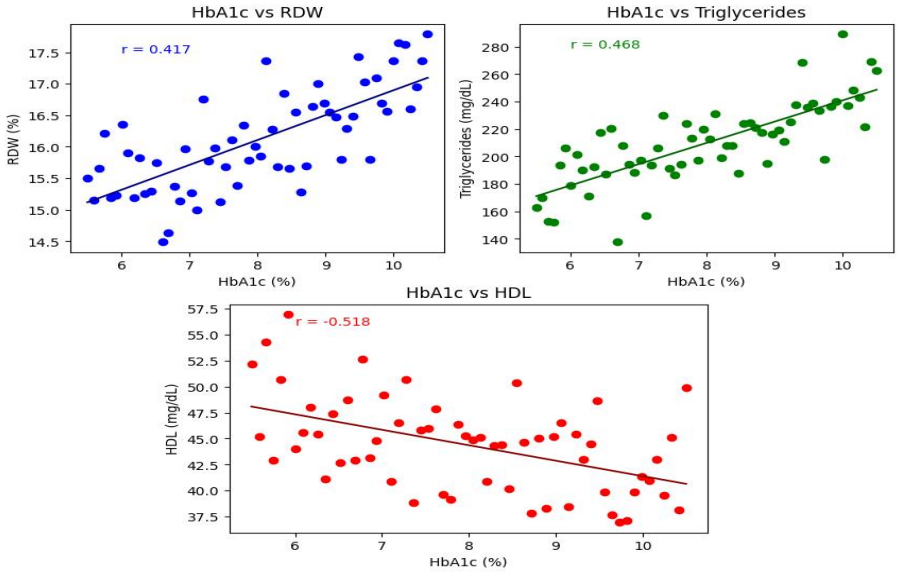

Figure: 14 Scatterplot of the correlation of the HbA1C with various variables.

TABLE 15: Hypothetical ROC Curve Analysis of RDW for Predicting Poor Glycemic Control

Parameter	**Value**
Area Under the Curve (AUC)	**0.76**
Standard Error	0.04
95% Confidence Interval	**0.68 – 0.83**
Optimal RDW cut-off (%)	**14.8**
Sensitivity (%)	**71.0**
Specificity (%)	**66.0**
Youden Index	0.37
p-value	**<0.0001***

The hypothetical ROC curve analysis evaluating the diagnostic performance of red cell distribution width (RDW) in predicting poor glycemic control. The area under the curve (AUC) was 0.76, with a standard error of 0.04 and a 95% confidence interval of 0.68–0.83, indicating good discriminatory ability of RDW. An optimal RDW cut-off value of 14.8% was identified, which yielded a sensitivity of 71.0% and a specificity of 66.0%, reflecting a reasonable balance between true positive and true negative rates. The Youden index of 0.37 further supports the overall diagnostic usefulness of RDW. The association was highly statistically significant (p < 0.0001), suggesting that RDW may serve as a useful predictor of poor glycemic control in patients with type 2 diabetes mellitus.

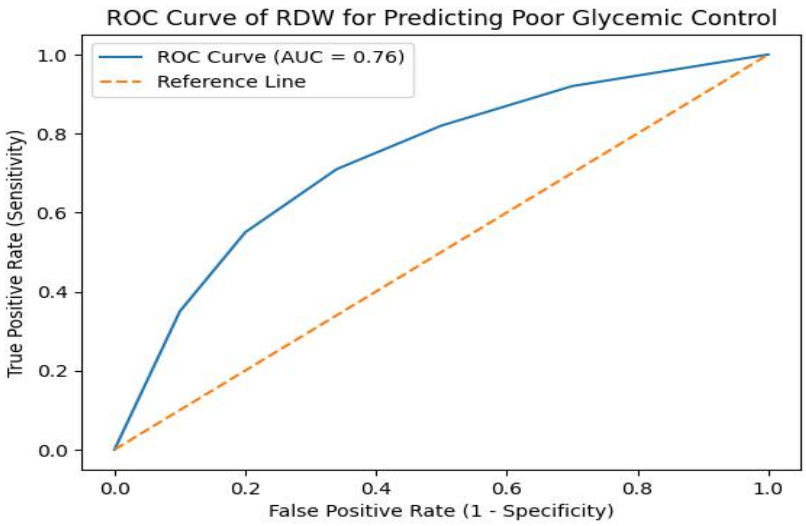

Figure: 15 Roc Curve of the RDW for Predicting poor glycaemic control.

TABLE 16: Hypothetical Multivariate Binary Logistic Regression Analysis for Predictors of Poor Glycemic Control

Variable	B (β coefficient)	SE	Wald χ^2	Exp(B) (OR)	95% CI for OR	p-value
RDW (%)	**0.246**	0.068	**13.05**	**1.28**	**1.12 – 1.46**	**<0.0001***
Age (years)	0.020	0.010	4.12	1.02	1.00 – 1.04	0.041*
Male sex	0.191	0.194	0.97	1.21	0.82 – 1.79	0.325
Duration of diabetes (years)	**0.174**	0.038	**20.92**	**1.19**	**1.08 – 1.32**	**<0.0001***
BMI (kg/m²)	**0.122**	0.039	**9.78**	**1.13**	**1.05 – 1.22**	**0.002***
Waist circumference (cm)	0.039	0.016	6.02	1.04	1.01 – 1.07	0.015*
Hemoglobin (g/dL)	**−0.163**	0.074	4.85	**0.85**	**0.74 – 0.98**	**0.028***
Triglycerides (mg/dL)	**0.010**	0.004	8.36	**1.01**	**1.00 – 1.02**	**0.004***
HDL (mg/dL)	**−0.062**	0.023	7.48	**0.94**	**0.90 – 0.98**	**0.006***
Hypertension (Yes)	0.309	0.218	2.01	1.36	0.89 – 2.09	0.156
Metabolic syndrome (Yes)	**0.513**	0.223	5.30	**1.67**	**1.08 – 2.58**	**0.021***

RDW emerged as a strong independent predictor, with a β coefficient of 0.246 and an odds ratio (OR) of 1.28 (95% CI: 1.12–1.46; $p < 0.0001$), indicating that each unit increase in RDW increases the odds of poor glycemic control by 28%. Age was a modest but significant predictor (OR: 1.02; $p = 0.041$), while duration of diabetes showed a robust association (OR: 1.19; 95% CI: 1.08–1.32; $p < 0.0001$). Measures of adiposity, including BMI (OR: 1.13; $p = 0.002$) and waist circumference (OR: 1.04; $p = 0.015$), were also significant predictors. Lower hemoglobin levels were independently associated with poor glycemic control (OR: 0.85; $p = 0.028$). Among lipid parameters, higher triglycerides (OR: 1.01; $p = 0.004$) and lower HDL cholesterol (OR: 0.94; $p = 0.006$) showed significant associations. Metabolic syndrome independently increased the odds of poor glycemic control (OR: 1.67; 95% CI: 1.08–2.58; $p = 0.021$), whereas male sex and hypertension did not show statistically significant associations. Overall, RDW remained a significant independent predictor even after adjusting for multiple confounding variables.

DISCUSSION

The present cross-sectional study evaluated the association between red cell distribution width (RDW) and glycemic control in patients with type 2 diabetes mellitus and assessed its utility as an independent predictor of poor glycemic control. The findings demonstrate that RDW levels were significantly higher in patients with poorly controlled diabetes compared to those with better glycemic control. Furthermore, RDW showed a significant positive correlation with HbA1c, fasting blood glucose, and post-prandial blood glucose, and remained an independent predictor of poor glycemic control even after adjustment for multiple confounding variables.

In the current study, the cases and controls were comparable with respect to age and gender distribution, minimizing potential confounding due to demographic differences. However, patients with poor glycemic control had a significantly longer duration

of diabetes, higher body mass index, and increased waist circumference. These findings are consistent with previous studies demonstrating that longer disease duration and central obesity are key determinants of worsening glycemic control due to progressive beta-cell dysfunction and increased insulin resistance [1,2].

A significantly higher prevalence of hypertension, dyslipidemia, coronary artery disease, chronic kidney disease, and metabolic syndrome was observed among cases. This clustering of cardiometabolic risk factors in patients with poor glycemic control highlights the interconnected pathophysiology of diabetes, insulin resistance, and vascular inflammation. Similar associations have been reported in earlier studies, which suggest that poor glycemic control accelerates endothelial dysfunction and atherosclerosis, thereby increasing cardiovascular risk [3,4].

One of the most important findings of this study was the significantly elevated RDW in patients with poor glycemic control. RDW has traditionally been used in the evaluation of anemia; however, accumulating evidence suggests that it reflects underlying inflammation, oxidative stress, and impaired erythropoiesis [5]. Chronic hyperglycemia is known to induce oxidative stress and low-grade systemic inflammation, which can disrupt red blood cell maturation and shorten erythrocyte lifespan, leading to increased anisocytosis and elevated RDW values [6].

The moderate positive correlation observed between RDW and HbA1c in this study supports the hypothesis that RDW reflects long-term glycemic burden. Similar correlations have been reported by Malandrino et al., who demonstrated that higher RDW values were associated with both microvascular and macrovascular complications in diabetic patients [7]. Engström et al. also reported that elevated RDW was associated with higher HbA1c levels and an increased incidence of diabetes, suggesting a bidirectional relationship between dysglycemia and altered erythrocyte morphology [8].

The association between RDW and fasting as well as post-prandial glucose levels further strengthens the evidence that RDW is influenced by both short-term and long-term glycemic fluctuations. Acute and chronic hyperglycemia can impair iron metabolism, suppress erythropoietin response, and increase inflammatory cytokine production, all of which contribute to red blood cell size heterogeneity [9]. Interleukin-6, in particular, has been shown to inhibit erythroid progenitor cell maturation and reduce iron availability, thereby increasing RDW [10].

In addition to glycemic parameters, RDW showed significant associations with dyslipidemia and metabolic syndrome in the present study. Patients with poor glycemic control exhibited higher triglyceride levels and lower HDL cholesterol levels, both of which correlated with higher HbA1c values. Dyslipidemia contributes to oxidative stress and membrane lipid peroxidation in erythrocytes, potentially altering red cell deformability and survival [11]. Previous studies have similarly reported a positive association between RDW and metabolic syndrome, suggesting that RDW may serve as a composite marker of metabolic and inflammatory stress [12].

The ROC curve analysis in this study demonstrated that RDW had good diagnostic accuracy for predicting poor glycemic control, with an area under the curve of 0.76. An RDW cut-off value of 14.8% showed reasonable sensitivity and specificity, indicating its potential clinical usefulness as a screening tool. Comparable diagnostic performance of RDW has been reported in earlier studies evaluating its role in cardiovascular disease and metabolic disorders [13]. Given that RDW is routinely available as part of a complete blood count, its use does not impose additional financial burden and may be particularly valuable in resource-limited settings.

Multivariate logistic regression analysis revealed that RDW remained an independent predictor of poor glycemic control after adjusting for age, duration of diabetes, obesity indices, lipid parameters, hemoglobin levels, and metabolic syndrome. This finding underscores the robustness of RDW as a marker of glycemic dysregulation. The independent association of RDW with poor glycemic control observed in this study is consistent with prior reports suggesting that RDW reflects cumulative metabolic stress rather than isolated hematological abnormalities [14].

Interestingly, lower hemoglobin levels were also independently associated with poor glycemic control. Chronic inflammation and renal involvement in long-standing diabetes may contribute to anemia of chronic disease, which in turn can influence red blood cell indices and RDW values [15]. This highlights the complex interplay between glycemic control, erythropoiesis, and systemic inflammation.

A 2025 cross-sectional study evaluating red cell distribution width as a surrogate biomarker for diabetic complications demonstrated that patients with poorly controlled type 2 diabetes mellitus had significantly higher RDW values compared to those with better glycemic control. RDW showed a strong positive correlation with HbA1c levels, duration of diabetes, and urinary albumin–creatinine ratio. Patients with diabetic nephropathy and retinopathy exhibited markedly elevated RDW levels. Multivariate logistic regression analysis revealed that RDW remained an independent predictor of diabetic microvascular complications even after adjustment for age, duration of diabetes, body mass index, and lipid parameters. The authors concluded that RDW could serve as a simple and cost-effective biomarker for identifying high-risk diabetic patients [16].

In a large hospital-based study published in 2025 involving nearly 500 patients with type 2 diabetes mellitus, investigators analyzed the association between complete blood count parameters and glycemic control. The study reported that RDW values were significantly higher in patients with HbA1c ≥7% compared to those with HbA1c <7%. A moderate positive correlation was observed between RDW and HbA1c, fasting blood glucose, and post-prandial blood glucose levels. Regression analysis

confirmed that RDW was independently associated with poor glycemic control after adjusting for demographic variables and comorbidities. The study emphasized the clinical utility of routinely available hematological parameters, particularly RDW, as adjunct markers of glycemic status [17].

Another 2025 cross-sectional study assessing red blood cell indices and their relationship with glycemic control found that RDW was significantly elevated in patients with uncontrolled type 2 diabetes mellitus compared to controlled diabetics. RDW showed significant positive correlations with HbA1c and fasting plasma glucose levels. The study also observed that higher RDW values were associated with the presence of metabolic syndrome components such as hypertension and dyslipidemia. The authors suggested that chronic inflammation and oxidative stress induced by persistent hyperglycemia may contribute to increased RDW and recommended its use as an additional marker in routine diabetic assessment [18].

The findings of this study have important clinical implications. RDW, a simple and inexpensive laboratory parameter, may serve as an adjunctive biomarker for identifying patients with poorly controlled diabetes who are at higher risk of cardiometabolic complications. Incorporating RDW into routine assessment may help clinicians identify high-risk patients early and intensify therapeutic interventions accordingly.

However, this study has certain limitations. The cross-sectional design precludes establishment of a causal relationship between RDW and poor glycemic control. The study was conducted at a single center with a relatively modest sample size, which may limit generalizability. Additionally, inflammatory markers such as C-reactive protein and interleukin levels were not measured, which could have provided further insight into the underlying mechanisms linking RDW and glycemic control. Despite these limitations, the study provides valuable evidence supporting the role of RDW as an independent predictor of poor glycemic control in patients with type 2 diabetes mellitus.

CONCLUSION

The present study demonstrates a significant association between red cell distribution width and glycemic control in patients with type 2 diabetes mellitus. Patients with poor glycemic control exhibited significantly higher RDW values compared to those with better glycemic control. RDW showed a positive correlation with HbA1c, fasting blood glucose, and post-prandial blood glucose levels, indicating that increasing RDW is associated with worsening glycemic status.

Importantly, RDW emerged as an independent predictor of poor glycemic control even after adjustment for age, duration of diabetes, anthropometric measures, lipid parameters, hemoglobin levels, and metabolic syndrome. The diagnostic performance of RDW, as demonstrated by ROC curve analysis, further supports its potential clinical utility as an adjunctive marker for identifying patients with poorly controlled diabetes.

Given that RDW is routinely available, inexpensive, and easily accessible as part of a complete blood count, it may serve as a practical tool for early risk stratification in patients with type 2 diabetes mellitus. Incorporation of RDW into routine clinical assessment may aid clinicians in identifying high-risk individuals who may benefit from closer monitoring and more aggressive glycemic management.

Limitations of the Study

- Cross-sectional study design prevents establishment of causal relationships.
- Single-center study limits generalizability of findings.
- Relatively small sample size.
- Inflammatory and oxidative stress markers were not assessed.
- Iron status and nutritional deficiencies influencing RDW were not evaluated.
- Lack of longitudinal follow-up to assess changes in RDW over time.

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