Biomedical and Biopharmaceutical Research
Abbreviation: Biomed. Biopharm. Res.
Volume: 22 | Issue: 02 | Year: 2025
Page Number: 910-919

COMPARISION OF THE COAGULATION PROFILE IN TYPE 2 DIABETES MELLITUS PATIENTS WITH GOOD GLYCAEMIC CONTROL AND POOR GLYCAEMIC CONTROL

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Article Received: 23-07-2025
Article Accepted: 25-08-2025

ABSTRACT

Type 2 diabetes significantly impacts the hemostatic system, fostering a prothrombotic state that accelerates atherosclerosis and heightens cardiovascular risk. These disruptions affect various components of coagulation, including platelet activity, clotting factors, natural anticoagulants, and the fibrinolytic pathway. In a comparative analysis involving 84 individuals with Type 2 diabetes, researchers evaluated coagulation patterns in two equally sized groups based on glycemic control those with HbA1c below 7% (well-controlled) and those with HbA1c equal to or above 7% (poorly controlled). The findings revealed meaningful differences in coagulation parameters between the groups. Participants with inadequate glycemic control exhibited longer prothrombin time (14.24 vs. 13.69 seconds; p=0.042) and activated partial thromboplastin time (33.28 vs. 32.32 seconds; p=0.001), alongside significantly increased D-dimer levels (649.45 vs. 560.43 ng/mL; p=0.001). These abnormalities were accompanied by markedly elevated urinary albumin (59.67 vs. 13.38 mg/dL; p=0.001) and higher rates of glycosuria (88.1% vs. 40.5%; p=0.001), indicating more advanced diabetic complications in the poorly controlled group. Notably, demographic characteristics such as age, sex, and hematological profiles were comparable across groups, eliminating confounding influences and underscoring glycemic control as the key variable. The co-existence of prolonged coagulation times with elevated D-dimer points to a hemostatic imbalance where clotting is simultaneously inefficient and hyperactive. Furthermore, the correlation with nephropathy indicators like albuminuria and glycosuria highlights the wide-reaching, interconnected nature of diabetes-related systemic damage. Ultimately, the study suggests that these coagulation anomalies might serve as novel biomarkers for cardiovascular risk in diabetic individuals. Most importantly, the results stress the vital role of consistent glycemic management not only to ward off classic diabetes complications but also to maintain balanced coagulation and reduce cardiovascular risk.

Keywords: Coagulation, Type 2 Diabetes Mellitus, Hemostasis, Glycated Haemoglobin, Glycaemic Control, HbA1c.

INTRODUCTION

Diabetes mellitus (DM) is a long-term, complex metabolic condition marked by elevated blood glucose levels due to impaired insulin production, reduced insulin sensitivity, or both. It represents one of the most critical health threats globally in the modern era. The International Diabetes Federation (IDF) estimates that by 2035, the worldwide number of

diabetes cases could rise to 592 million.[1] Persistent hyperglycemia is a central factor in the development of both small and large blood vessel complications such as diabetic eye disease (retinopathy), kidney damage (nephropathy), nerve impairment (neuropathy) and cardiovascular issues. These health challenges not only compromise an individual's quality of life but also place enormous strain on healthcare systems and contribute to early disability and death.[2]

Recent research underscores the impact of disrupted blood clotting mechanisms and elevated thrombotic risk in the development of diabetes-related complications. People with type 2 diabetes mellitus (T2DM) often display a state of hypercoagulability, which influences both large and small blood vessels and contributes to disease progression. This tendency toward excessive clotting arises from multiple factors: heightened platelet responsiveness, elevated fibrinogen concentrations, increased thrombin activity, and compromised breakdown of clots (fibrinolysis).[2,3] Chronic high blood sugar is a key player in triggering and maintaining these coagulation irregularities. Diabetic platelets become more reactive to substances like adenosine diphosphate (ADP) and thrombin, leading to stronger tendencies for clumping and sticking together. Higher levels of plasma fibrinogen have also been observed, which thickens the blood and encourages clot formation.[4] To assess clotting function, prothrombin time (PT) and activated partial thromboplastin time (aPTT) are commonly used evaluating the external and internal pathways of the coagulation cascade, respectively. Research suggests that people with diabetes may experience shortened PT and aPTT, indicating a quicker initiation of clotting processes.[5]

The connection between blood sugar management and clotting factors is still being actively explored. Although diabetes is known to affect hemostatic balance, routine testing for coagulation abnormalities is rarely part of standard care for diabetic individuals. In most cases, patients are only assessed after experiencing serious thrombotic complications such as stroke, heart attack**,** or deep vein thrombosis, by which point damage to organs may already be irreversible.[3] This highlights the importance of detecting blood clotting irregularities during the early or asymptomatic stages of diabetes. Monitoring these changes in correlation with glycemic control may offer valuable insights for risk assessment and enable timely preventive interventions**.**

In addition, research directly comparing the blood clotting profiles of individuals with well-managed versus poorly controlled diabetes remains limited, and even fewer studies have included healthy participants to provide baseline data. To address this gap, the current study aims to assess and contrast the coagulation characteristics of patients with type 2 diabetes, categorized by the quality of their glycemic control. Key indicators such as prothrombin time (PT), activated partial thromboplastin time (aPTT), and other relevant biomarkers were measured to determine whether notable hemostatic variations exist between these diabetic subgroups and non-diabetic individuals. By exploring these comparisons, the study seeks to clarify how elevated blood glucose levels may influence the coagulation process, and whether these markers can serve as early warning signs for increased risk of thrombotic complications.[6]

METHODOLOGY

This investigation followed a hospital-based comparative observational design and was conducted within the Department of Pathology at Government Medical College and its associated hospital group in Kota**.** The study spanned 12 months**,** from October 2023 to

September 2024, and was undertaken after receiving approvals from both the Institutional Research Review Board and the Institutional Ethics Committee. Participants were recruited until the required sample size was reached, resulting in a total of 84 subjects, with 42 individuals assigned to each group.

The sample size was calculated using the formula: $N = [2(\alpha^2 + \beta^2)(S1^2 + S2^2)] / (M1 - M2)^2$ $N=[2(\alpha^2+\beta^2)\ (S1^2+S2^2)]\ (M1\text{-}M2)^{\ 2}$ Where S1=29.63 S2=34.61 M1=155.57 M2=12.19 S: Standard Deviation M: Mean N= 42 in each group.

This research included individuals diagnosed with type 2 diabetes mellitus (T2DM) who were undergoing antidiabetic therapy and were either attending outpatient services or admitted to the hospital. It followed a comparative observational study design.

Inclusion Criteria

- **Group 1:** T2DM patients receiving antidiabetic treatment with HbA1c < 7%, indicating good glycemic control
- **Group 2:** T2DM patients receiving antidiabetic treatment with HbA1c ≥ 7%, indicating poor glycemic control

Exclusion Criteria

- Patients diagnosed with septicemia, bleeding disorders, liver disease, malignancy, pregnancy, or those who were postoperative or on antiplatelet therapy
- Individuals with type 1 diabetes mellitus
- Patients taking anticoagulant medications

Sampling Technique

A convenience sampling approach was used to select eligible participants. A total of 84 subjects met the inclusion criteria, with 42 patients in each group.

Methodology

After obtaining informed consent in each participant's preferred language, the following steps were taken:

- Collection of detailed clinical history, including demographic details, duration of diabetes, current medications, and any comorbid conditions
- Venous blood samples (5 mL) were drawn under sterile conditions

Laboratory Assessments

Samples were tested for Platelet Count, Prothrombin Time (PT), Activated Partial Thromboplastin Time (aPTT), Glycated Hemoglobin (HbA1c), D-dimer Levels using standard protocols and equipment.

Statistical Analysis

Data were analyzed using SPSS version 21.0 (Chicago, Illinois, USA). Key procedures included:

- Univariate analysis, presented through tables, narratives, bar graphs, and pie charts
- Descriptive statistics: Frequencies and percentages for categorical variables; mean, standard deviation, and range for continuous data
- Independent t-tests for comparing continuous variables between groups
- Chi-square tests to examine associations in categorical data
- A p-value < 0.05 was set as the threshold for statistical significance

RESULTS

The average age of participants in Group I (well-controlled glycemia) was 51.55 ± 11.48 years, while Group II (poorly controlled glycemia) had a slightly younger mean age of 49.69

± 11.68 years. Median ages were 52.0 years for Group I and 49.5 years for Group II. The age distribution spanned 33 to 73 years in Group I and 28 to 82 years in Group II. Statistical analysis revealed no significant difference between the age profiles of the two groups (p = 0.465), indicating they were age-matched. In terms of age subgroups: Participants aged ≤40 years comprised 23.8% in both groups. In the 41–60 years bracket, Group II had a higher proportion (64.3%) compared to Group I (50.0%). For those above 60 years, Group I included more individuals (26.2%) than Group II (11.9%). These variations were not statistically significant (p = 0.223), suggesting age stratification was relatively similar. When evaluating gender distribution: Group I had a predominance of male participants (69.0%), while females made up 31.0%. In Group II, females represented a higher share (45.2%) and males 54.8%. The difference in gender composition between the groups was not statistically significant (p = 0.178), indicating gender did not influence the study outcomes..

Table 1: Distribution of demographic variables: age (years), age group, gender between both groups (Group I and Group II)

Demographic variables	**Group I**				**Group II**				**p-value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
Age (Years)	51.55±11.48	52.00	33.0	73.0	49.69±11.68	49.500	28.0	82.0	
			Mean±SD	**Median**	**Min**	**Max**			0.465 (NS)
Total			50.61±11.55	51.500	28.0	82.0			
Age group (Years)	**Group I**	**Count**	**%**	**Total**	**Group II**	**Count**	**%**	**Total**	**p-value**
	≤40 years	10	23.8%	42 (100%)	≤40 years	10	23.8%	42 (100%)	0.223 (NS)
	41-60 years	21	50.0%		41-60 years	27	64.3%		
	>60 years	11	26.2%		>60 years	5	11.9%		
Gender	**Group I**	**Count**	**%**	**Total**	**Group II**	**Count**	**%**	**Total**	**p-value**
	Male	29	69.0%	42 (100%)	**Male**	23	54.8%	42 (100%)	0.178 (NS)
	Female	13	31.0%		**Female**	19	45.2%		

Table 2: Comparision of clinical variables: FBS, HbA1c, urine albumin, glycosuria, RBC, WBC, platelets, Hb, Hematocrit, prothrombin, activated partial thromboplastin time (aPTT), d-dimer between both groups (Group I and Group II)

Clinical variables	**Group I**				**Group II**				**p-value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
FBS	196.1±95.21	157.000	81.0	438.0	177.67 ±69.16	163.500	90.0	414.0	
Total			**Mean±SD**	**Median**	**Min**	**Max**			0.001 (S)
			186.8±83.22	161.00	81.0	438.0			
HbA1c	**Group I**				**Group II**				**p-value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
	6.15±0.53	6.150	5.0	6.9	9.13±1.73	8.90	7.0	14.5	
Total			**Mean±SD**	**Median**	**Min**	**Max**			0.001 (S)
			7.64±1.97	6.95	5.0	14.5			
Urine alb	**Group I**				**Group II**				**p-value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
	13.38±1.98	14.2000	9.03	16.50	59.67±4.34	59.00	50.00	69.00	0.001 (S)
Total			**Mean±SD**	**Median**	**Min**	**Max**			
			36.52±23.52	33.25	9.03	69.00			
Glycosuria	**Group I**	**Count**	**%**		**Group II**	**Count**	**%**		**p- value**
Yes		17	40.5%			37	88.1%		0.001 (S)
No		25	59.5%			5	11.9%		
Total	42 (100%)				42 (100%)				
RBC	**Group I**				**Group II**				
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	**p- value**
	4.68±0.74	4.8400	2.94	5.80	4.90±0.70	4.86	3.70	6.75	0.159 (N.S)
Total			**Mean±SD**	**Median**	**Min**	**Max**			

			4.79±0.72	4.84	2.94	6.75			
WBC	**Group I**				**Group II**				**p- value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	0.855
	8.61±3.33	8.2200	4.19	19.70	8.50±2.08	8.64	4.17	12.92	(N.S)
Total			**Mean±SD**	**Median**	**Min**	**Max**			
			8.56±2.76	8.59	4.17	19.70			
Platelet	**Group I**				**Group II**				**p- value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	0.871
	285.43±91.27	290.500	109.0	467.0	282.26±87.46	264.50	145.0	562.0	(N.S)
			Mean±SD	**Median**	**Min**	**Max**			
Total			283.84±88.86	285.00	109.0	562.0			
Hb	**Group I**				**Group II**				**p- value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
	12.24±2.08	11.900	7.7	15.8	13.11 ±1.63	13.20	8.7	15.6	0.136
Total			**Mean±SD**	**Median**	**Min**	**Max**			(N.S)
			12.68±1.91	12.90	7.7	15.8			
Hematocrit	**Group I**				**Group II**				**p- value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
	37.55±5.89	36.6000	26.00	47.50	49.84 ±59.61	41.65	29.50	426.00	0.187
Total			**Mean±SD**	**Median**	**Min**	**Max**			(N.S)
			43.69±42.55	39.60	26.00	426.00			
Prothrombin time	**Group I**				**Group II**				**p- value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
	13.68±1.20	13.9800	11.25	15.89	14.24 ±1.25	14.26	11.32	16.55	0.042
Total			**Mean±SD**	**Median**	**Min**	**Max**			(S)
			13.96± 1.24	14.16	11.25	16.55			
aPTT	**Group I**				**Group II**				**p- value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
	32.32 ±1.11	32.3750	30.23	34.87	33.28±1.51	33.32	30.87	35.58	0.001 (S)
Total			**Mean±SD**	**Median**	**Min**	**Max**			
			32.80± 1.40	32.49	30.23	35.58			
d-dimer	**Group I**				**Group II**				**p- value**
	Mean±SD	**Median**	**Min**	**Max**	**Mean±SD**	**Median**	**Min**	**Max**	
	560.43±12.3	563.000	531.0	577.0	649.45±19.56	647.50	605.0	692.0	0.001 (S)
Total			**Mean±SD**	**Median**	**Min**	**Max**			
			604.94±47.6	591.00	531.0	692.0			

Surprisingly, despite being classified as having better glycemic control, Group I showed a higher mean fasting blood sugar (FBS) of 196.07 mg/dL compared to Group II at 177.67 mg/dL. This unexpected result was statistically significant ($p = 0.001$), suggesting the need for further clinical interpretation or verification of group assignments. Conversely, HbA1c levels were appropriately aligned with group definitions, Group I had a significantly lower mean HbA1c of 6.15% (range 5.0–6.9%), and while Group II averaged 9.13% (range 7.0–14.5%) ($p = 0.001$), affirming classification accuracy. Urine albumin levels, an indicator of diabetic nephropathy, were significantly elevated in Group II (59.67 mg/dL) compared to Group I (13.38 mg/dL) ($p = 0.001$), highlighting a strong correlation between poor glycemic control and albuminuria. Similarly, glycosuria was far more prevalent in Group II (88.1%) versus Group I (40.5%) ($p = 0.001$), while the absence of glycosuria was more common in Group I (59.5%) compared to Group II (11.9%), reinforcing the metabolic imbalance in Group II.

Red blood cell (RBC) counts were slightly higher in Group II (4.90 million/mm³) than in Group I (4.68 million/mm³), though the difference was not statistically significant ($p = 0.159$), indicating similar erythropoietin activity. White blood cell (WBC) counts also showed negligible variation: 8.62 $\times 10^3$/mm³ in Group I and 8.50 $\times 10^3$/mm³ in Group II ($p = 0.855$), suggesting no notable inflammatory or infectious conditions across groups. Platelet counts were comparable between the groups (Group I: 285.43 $\times 10^3$/mm³, Group II: 282.26

$\times 10^3/mm^3$, p = 0.871), and hemoglobin levels were slightly higher in Group II (13.11 g/dL) versus Group I (12.24 g/dL) without statistical significance (p = 0.136). Hematocrit levels followed a similar trend, with Group II averaging 49.84% and Group I 37.55%, but extreme variability in Group II (maximum = 426.0%) suggests potential outliers or data inconsistencies (p = 0.187).

Prothrombin time (PT) was significantly prolonged in Group II (14.24 seconds) compared to Group I (13.69 seconds, p = 0.042). Activated partial thromboplastin time (aPTT) was longer in Group II (33.28 seconds) versus Group I (32.32 seconds, p = 0.001), indicating a disturbance in the intrinsic pathway. D-dimer levels were also notably higher in Group II (649.45 ng/mL) than in Group I (560.43 ng/mL) with a highly significant p-value (0.001), suggesting increased clotting activity or enhanced fibrinolysis in poorly controlled diabetes.

DISCUSSION

Demographic and Baseline Overview

This study involved 84 individuals with type 2 diabetes mellitus (T2DM), equally divided into two groups according to their level of glycemic control. An analysis of demographic parameters offered key insights that helped contextualize the comparison of coagulation profiles.

Age Distribution and Its Significance

The average age of participants in Group I (good glycemic control) was 51.55 ± 11.48 years, while in Group II (poor glycemic control) it was 49.69 ± 11.68 years. This difference was not statistically significant (p = 0.465), ensuring that age did not act as a confounding factor when examining the effects of glycemic control on hemostasis. Balanced age distributions between the groups enhance the reliability of the comparative analysis, especially since age-related changes in clotting mechanisms can influence thrombotic risk. Further breakdown showed that, Group II had a greater number of participants in the 41–60 years age bracket (64.3% vs. 50.0%), Group I had more individuals aged over 60 years (26.2% vs. 11.9%).Although these trends might suggest younger individuals struggle more with glycemic regulation possibly due to lifestyle habits, medication adherence, or disease dynamics, the non-significant p-value (0.223) confirms this age variation did not skew the study outcomes. Findings echo observations from **Agarwal C et al**[7], which emphasized the importance of age parity in studies evaluating coagulation patterns in diabetics.

Gender Distribution and Physiological Considerations

Analysis of gender composition revealed a higher proportion of males in Group I (69.0%), suggesting a trend toward better glycemic control among men, a more balanced profile in Group II (54.8% males, 45.2% females). Despite this contrast, the difference was not statistically significant (p = 0.178). Still, the data raise considerations about the role gender might play in diabetes management and its impact on coagulation. Hormonal influences, such as the varying effects of estrogen, have been shown to both promote and inhibit clotting, depending on physiological conditions. These results align in part with findings by **Getu F et al**[8]**,** where female gender was associated with increased coagulation abnormalities. In their study, women comprised 52.1% of the sample and demonstrated a greater tendency toward hypercoagulability. Therefore, although not statistically conclusive in this analysis, the

greater number of females in the poorly controlled group may offer relevant clues into the gendered patterns of clotting disorders in diabetes.

Glycemic Control Parameters and Metabolic Markers

HbA1c Levels and Long-term Glycemic Control

The notable difference in HbA1c levels between the two study groups validated their classification. Group I had significantly lower values (6.15%) compared to Group II (9.13%, $p = 0.001$), representing an approximate 3% gap that holds clinical importance for assessing long-term glucose control and cardiovascular risk. Group I's HbA1c range (5.0–6.9%) reflects optimal to moderate glycemic regulation in accordance with modern diabetes guidelines, while Group II's range (7.0–14.5%) indicates varying levels of poor control, from borderline to severely uncontrolled. This contrast forms a critical basis for understanding variations in coagulation profiles. Long-standing hyperglycemia is associated with a prothrombotic state, driven by the formation of advanced glycation end products (AGEs), increased oxidative stress, and endothelial dysfunction. Comparable findings were reported by **Sherin B et al**[9], who used similar HbA1c thresholds and observed significant disparities in coagulation measures between well-controlled and poorly controlled diabetic patients. An intriguing observation was the higher fasting blood sugar (FBS) in Group I (196.07 mg/dL) compared to Group II (177.67 mg/dL), despite Group I being classified as better controlled. This discrepancy, which was statistically significant ($p = 0.001$), contradicts conventional trends where FBS aligns closely with HbA1c. Prior studies such as those by **Agarwal C et al**[7] and **Mariappan A et al**[10] showed consistent associations between fasting glucose and overall glycemic status, making this divergence a compelling area for further study and clinical review. Analysis of urine albumin levels revealed a pronounced elevation in Group II (59.67 mg/dL) versus Group I (13.38 mg/dL, $p = 0.001$), highlighting a strong link between poor glycemic control and kidney damage progression. Albuminuria is widely regarded as an early and reliable marker of diabetic nephropathy. The significantly greater concentration in Group II nearly 4.5 times higher not only suggests the onset of nephropathy but may reflect advanced renal impairment in these patients. Typically, normal albumin excretion is below 30 mg/day or 20 mg/L in spot urine samples. The average of 59.67 mg/dL in Group II clearly indicates macroalbuminuria, which correlates with both established kidney disease and elevated cardiovascular risk.

Diabetic nephropathy significantly contributes to increased clotting risk through mechanisms such as disrupted protein synthesis, imbalances in mineral metabolism, and persistent inflammation. As the kidneys are central to both the production and elimination of various coagulation factors, kidney dysfunction serves as a notable confounding variable in studies assessing hemostatic changes. The analysis of urinary glucose strongly reinforced the glycemic classification, with 88.1% of Group II (poor control) displaying glycosuria compared to 40.5% of Group I (good control), a difference that was statistically significant ($p = 0.001$). This supports the observed disparities in HbA1c and highlights recurrent hyperglycemia among poorly controlled individuals. Glycosuria typically arises when blood glucose surpasses the renal threshold (~180 mg/dL), reflecting frequent spikes in blood sugar.

Interestingly, glycosuria also appeared in 40.5% of well-controlled patients, possibly due to post-meal glucose surges, individual variations in renal threshold, or transient factors like illness, medication changes, or dietary indulgence. The combination of elevated albuminuria and glycosuria in Group II points toward more advanced metabolic disturbances, including

potential vascular damage, inflammatory processes, and protein handling anomalies all of which may contribute to clotting irregularities.

When comparing RBC counts, both groups showed similar results (4.68 vs. 4.90 million/mm³, $p = 0.159$), suggesting equivalent red blood cell production despite differences in glycemic control. This consistency indicates that coagulation differences are not likely driven by variations in red cell mass or related hematologic conditions. The slightly higher RBC count in Group II may reflect adaptive physiological responses to persistent hyperglycemia such as elevated erythropoietin triggered by tissue hypoxia, fluid shifts due to osmotic diuresis, or adjustments compensating for reduced oxygen efficiency linked to glycosylated hemoglobin. Hemoglobin levels (Group I: 12.24 g/dL; Group II: 13.11 g/dL; $p = 0.136$) were similarly unaffected, further eliminating anemia as a possible confounding factor.

Platelet counts were nearly identical between both groups (285.43 vs. 282.26 $\times 10^3$/mm³, $p = 0.871$), indicating that the number of platelets does not account for observed differences in coagulation profiles. This highlights that diabetic coagulation disorders are more likely driven by functional changes in platelet behavior such as altered adhesion, aggregation, and cross-talk with clotting factors rather than simple quantitative deviations. These findings are consistent with research by **Ephraim RK et al[11]**, which found no significant platelet count difference between diabetic and non-diabetic populations. However, it's important to note that while their study compared diabetics to healthy controls, the current analysis focuses on differences among diabetics with varying degrees of glycemic regulation.

The white blood cell (WBC) count findings 8.62 vs. 8.50 $\times 10^3$/mm³, $p = 0.855$ indicate a similar inflammatory or infectious status between the two groups at the time of analysis. This similarity helps ensure that differences in coagulation parameters are likely due to chronic metabolic dysfunction from poorly managed diabetes rather than acute inflammatory processes. It's worth noting, however, that subtle chronic inflammation, commonly seen in diabetes, may not manifest through elevated WBC counts but could still influence coagulation via cytokines or other inflammatory mediators. Extrinsic Pathway Assessment via Prothrombin Time revealed a significant increase in PT among poorly controlled diabetics (14.24 vs. 13.69 seconds, $p = 0.042$), indicating potential dysfunction in the extrinsic coagulation pathway. This pathway involves several clotting factors VII, X, V, II (prothrombin), and fibrinogen suggesting that hyperglycemia may affect their synthesis or function. Potential underlying causes include:

- Liver impairment due to chronic hyperglycemia, affecting coagulation factor production
- Glycation of proteins, altering clotting factor structure and reducing activity
- Vitamin K metabolism changes, impacting synthesis of key clotting components
- Chronic inflammation, which can influence production and degradation rates of coagulation proteins

These results differ from some prior studies, such as **Sherin B et al[9]**, which observed no PT differences. However, they align with **Ephraim RK et al[11]**, who reported PT prolongation when comparing diabetics to non-diabetics indicating population differences might explain the divergence.

A clear and statistically significant prolongation of aPTT was observed in Group II (33.28 vs. 32.32 seconds, $p = 0.001$), highlighting disruptions in the intrinsic coagulation pathway. This pathway involves clotting factors XII, XI, IX, VIII, X, V, II, and fibrinogen. Possible mechanisms include:

- Factor deficiencies due to reduced synthesis or higher consumption
- Presence of inhibitors or elevated natural anticoagulants
- Accumulation of heparin-like substances
- Renal dysfunction, as seen in Group II, may contribute via uremic toxins affecting coagulation

This finding is consistent with work by **Sherin B et al[9] and Agarwal C et al[7]**, although it contrasts with **Ephraim RK et al[11]** work, which showed shortened aPTT in diabetic individuals likely due to differences in study design and comparison groups.

D-dimer values were notably elevated in Group II (649.45 vs. 560.43 ng/mL, $p = 0.001$), suggesting heightened fibrinolytic or thrombotic activity in those with poor glycemic control. Elevated D-dimer levels may arise from:

- Increased thrombin and fibrin generation, indicating a hypercoagulable state
- Enhanced fibrinolysis, activated as a countermeasure
- Subclinical thrombosis, reflecting ongoing microvascular clot formation and breakdown
- Chronic inflammation, which can stimulate both clot formation and dissolution

Although **Sherin B et al[9]** did not observe significant changes in D-dimer between glycemic groups, such discrepancies could stem from differences in assay methods or clinical characteristics of the study populations.

The combined findings of prolonged PT and aPTT along with elevated D-dimer levels in poorly controlled diabetic individuals suggest a complex hemostatic imbalance one that includes both slowed coagulation pathways and intensified fibrinolytic activity. This imbalance has important clinical implications, potentially contributing to higher cardiovascular risk, advancing diabetic complications, and increased mortality in this population.

CONCLUSION

This study clearly establishes that inadequate glycemic control in individuals with type 2 diabetes mellitus is linked to marked disruptions in coagulation dynamics, demonstrated by prolonged clotting times and elevated levels of fibrinolytic markers. The simultaneous presence of extended PT and aPTT alongside increased D-dimer concentrations points to a multifaceted coagulation disorder characterized by both diminished clotting efficiency and heightened thrombotic activity. While certain findings resonate with existing literature, this study also highlights distinctive patterns that likely stem from the prolonged metabolic disturbances induced by chronic hyperglycemia. Furthermore, the strong relationship between poor glycemic control and markers of diabetic nephropathy, such as albuminuria and glycosuria, reinforces the widespread nature of diabetes-related complications and their intricate physiological interconnections. Importantly, the coagulation irregularities observed may hold value as additional biomarkers for evaluating cardiovascular risk in diabetic

populations providing insight beyond conventional metrics like blood glucose and HbA1c. Ultimately, these results underscore the vital importance of sustaining optimal glycemic control, not only to curb typical diabetic complications but also to support balanced hemostatic function and mitigate cardiovascular risk.

ACKNOWLEDGEMENT

The authors wish to acknowledge the contribution of **Dr. Shailendra Vashistha** (Assistant Professor, Dept of IHTM & HLA Lab, GMC, Kota) and the **VAssist Research** Team (www.thevassist.com) for their contribution in manuscript editing and submission process.

Conflict of interest: None.

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