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Association of Coagulation Profile with Microvascular Complications In Type II Diabetes Mellitus

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Abstract

Background: Type II Diabetes Mellitus (T2DM) is increasingly recognised as a prothrombotic state in which alterations in the coagulation cascade contribute to the pathogenesis of microvascular complications — diabetic nephropathy, neuropathy, and retinopathy — through microthrombus formation and impaired fibrinolysis within the glomerular, neural and retinal microvasculature.

Objective: To evaluate the association between coagulation profile (prothrombin time [PT], activated partial thromboplastin time [aPTT], and fibrinogen) and microvascular complications in patients with T2DM.

Methods: This observational cross-sectional study was conducted over 18 months at Gandhi Medical College and Hamidia Hospital, Bhopal. A total of 128 patients with T2DM were enrolled and stratified into four groups — Diabetic Nephropathy (n=35), Diabetic Neuropathy (n=17), Diabetic Retinopathy (n=12), and Diabetes without Complications (n=64) — based on urinary albumin-creatinine ratio, standardised nerve conduction test and fundoscopic screening. PT, aPTT, and fibrinogen (Clauss method) were measured on an automated coagulation analyser and compared across groups using one-way ANOVA and chi-square tests.

Results: The overall cohort showed a tendency toward hypercoagulability, with 54.7% having shortened PT, 60.2% shortened aPTT, and 53.9% elevated fibrinogen (mean 4.32 ± 1.20 g/L). Fibrinogen was significantly elevated in all three complication groups (Nephropathy 5.06 ± 1.23 g/L; Neuropathy 5.33 ± 1.03 g/L; Retinopathy 5.18 ± 1.37 g/L) compared with controls (4.08 ± 1.14 g/L;

F=9.133, p<0.001); all patients with complications had fibrinogen >4.0 g/L versus only 7.8% of controls (χ^2=104.182, p<0.001). Mean PT and aPTT did not differ significantly across groups (p=0.716 and p=0.636, respectively). Biochemical parameters, including HbA1c, showed no significant inter-group differences (all p>0.05).

Conclusion: Fibrinogen demonstrated a significant and independent association with microvascular complications in patients with T2DM, highlighting hyperfibrinogenaemia as a potential marker of microvascular complication burden. These findings support the inclusion of fibrinogen assessment in routine coagulation profiling as a useful adjunct for identifying patients at increased risk of diabetic microvascular complications.

Keywords: Type II Diabetes Mellitus, Coagulation Profile, Fibrinogen, PT (Prothrombin Time), aPTT (Activated Partial Thromboplastin Time), Microvascular Complications, Diabetic Nephropathy, Diabetic Retinopathy, Diabetic Neuropathy.

Introduction

Diabetes mellitus is one of the most significant global health challenges of the twenty-first century, with the International Diabetes Federation estimating approximately 537 million adults aged 20–79 years living with the disease worldwide, a figure projected to rise to 783 million by 2045.[1] Type II Diabetes Mellitus (T2DM) accounts for nearly 90–95% of all cases and is characterised by progressive insulin resistance and relative insulin deficiency arising from a complex interplay of genetic predisposition, obesity, physical inactivity, and ageing. The resulting chronic hyperglycaemic state predisposes patients to a wide spectrum of macrovascular and microvascular complications, with the latter — diabetic retinopathy, nephropathy, and neuropathy — being major contributors to morbidity and mortality.[2] Diabetic retinopathy remains the leading cause of blindness among working-age adults, diabetic nephropathy is the foremost cause of end-stage renal disease worldwide, and diabetic neuropathy affects up to half of all diabetic individuals, predisposing them to foot ulceration and amputation.

While the pathogenesis of these complications has traditionally been attributed to metabolic derangements such as advanced glycation end-product formation, polyol pathway activation, and protein kinase C activation, accumulating evidence over the past two decades indicates that diabetes mellitus also represents a distinct prothrombotic state, with haemostatic abnormalities playing a crucial role in disease progression.[3] Patients with T2DM demonstrate a hypercoagulable phenotype characterised by increased platelet reactivity, elevated procoagulant factors (fibrinogen, factor VII, factor VIII, and von Willebrand factor), impaired fibrinolysis due to elevated plasminogen activator inhibitor-1 (PAI-1), and endothelial dysfunction with loss of the endothelium's normal anticoagulant properties.[4] This relationship between coagulation abnormalities and microvascular damage appears bidirectional: hypercoagulability promotes microthrombus formation within retinal, glomerular, and neural capillaries, while the resulting tissue injury further amplifies procoagulant activity, sustaining a self-perpetuating cycle of vascular injury, as demonstrated by histopathological evidence of fibrin deposits and platelet aggregates in the microvasculature of diabetic patients with retinopathy and nephropathy.[5]

Coagulation activation markers such as D-dimer and thrombin-antithrombin complexes have been correlated with the severity of diabetic retinopathy,[6] while elevated

fibrinogen, factor VIII, and von Willebrand factor levels correlate with proteinuria and glomerular filtration rate decline in diabetic nephropathy.[7] Evidence for a haemostatic contribution to diabetic neuropathy, mediated through ischaemic injury of the vasa nervorum, is comparatively less extensive but growing.[8] These insights carry therapeutic relevance, as antiplatelet and anticoagulant strategies have been explored as adjuncts to conventional glycaemic, blood pressure, and lipid-directed management, although the evidence for benefit remains inconsistent and must be weighed against bleeding risk.[9] Given the persisting uncertainty regarding the relative contribution of individual coagulation parameters to specific microvascular complications, and the potential utility of coagulation profiling as a biomarker for risk stratification,[10] the present study was undertaken to systematically evaluate the association between coagulation profile — prothrombin time, activated partial thromboplastin time, and fibrinogen — and the presence of microvascular complications in patients with Type II Diabetes Mellitus.

Methodology

This observational cross-sectional study was conducted over 18 months (May 2024–October 2025) in the Department of Pathology in collaboration with the Department of Medicine, Gandhi Medical College and associated Hamidia Hospital, Bhopal, following approval from the Institutional Ethics Committee. A total of 128 patients with established Type II Diabetes Mellitus (fasting glucose ≥126 mg/dL, postprandial glucose ≥200 mg/dL, HbA1c ≥6.5%) were enrolled after written informed consent, using a sample size calculated at 95% confidence with an anticipated prevalence of 14% and a 6% dropout allowance. Patients with thrombocytosis, venous thromboembolism, inherited coagulation disorders, malignancy, pregnancy, recent surgery, or those on anticoagulant therapy were excluded.

All enrolled patients underwent standardised screening for microvascular complications, comprising nerve conduction studies for diabetic neuropathy, urinary albumin-to-creatinine ratio (UACR) for diabetic nephropathy (microalbuminuria: 30–300 mg/g; macroalbuminuria: >300 mg/g), and dilated fundoscopic examination for diabetic retinopathy, before patients were stratified into four groups: Diabetic Nephropathy, Diabetic Neuropathy, Diabetic Retinopathy, and Diabetes without Complications (controls). Venous blood was collected under aseptic precautions into sodium citrate vacutainers (9:1 blood-to-anticoagulant ratio) for coagulation studies, and into EDTA/plain vacutainers for biochemical and HbA1c estimation. Prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen (Clauss method) were measured on the Auto Coaglyzer XL 1000 C using photo-optical clot detection, following a three-level quality-control protocol. Biochemical parameters — liver function tests, renal function tests, and lipid profile — were estimated on the Beckman Coulter AU 5800 analyser using standard spectrophotometric and enzymatic methods.

Data were compiled in Microsoft Excel and analysed using SPSS and GraphPad Prism. Continuous variables were expressed as mean ± standard deviation and compared across groups using one-way ANOVA; categorical variables were expressed as frequencies and percentages and compared using the chi-square test. Correlation between coagulation parameters and microvascular complications was assessed using Pearson's or Spearman's correlation coefficient, as appropriate, with multivariate analysis performed to

adjust for confounders. A p-value <0.05 was considered statistically significant.

Results

A total of 128 patients with Type II Diabetes Mellitus were evaluated for coagulation parameters (PT, aPTT, fibrinogen) and relevant biochemical indices, and were stratified into four diagnostic groups — Diabetic Nephropathy, Diabetic Neuropathy, Diabetic Retinopathy, and Diabetes without Complications — based on standardised nerve conduction, UACR, and fundoscopic screening.

Table 1: Baseline Demographic Characteristics and Coagulation Parameter Distribution of Study Participants (n=128)

Variable	Category	Frequency (n)	Percent (%)
Age	31–40 years	9	7.0
	41–50 years	30	23.4
	51–60 years	55	43.0
	≥61 years	34	26.6
Sex	Female	61	47.7
	Male	67	52.3
PT	≤11.9 s	70	54.7
	12.0–16.5 s	50	39.1
	>16.5 s	8	6.3
aPTT	≤26.9 s	77	60.2
	27.0–42.5 s	50	39.1
	>42.5 s	1	0.8
Fibrinogen	<2.0 g/L	1	0.8
	2.0–4.0 g/L	58	45.3
	>4.0 g/L	69	53.9
Diagnostic Group	Diabetic Nephropathy	35	27.3
	Diabetic Neuropathy	17	13.3
	Diabetic Retinopathy	12	9.4
	Diabetes without Complications	64	50.0

The cohort was predominantly middle-aged and older, with 69.6% aged above 50 years, and showed a near-equal sex distribution, minimising sex- and age-related confounding (Table 1). More than half the participants had shortened PT (54.7%) and aPTT (60.2%), and 53.9% had elevated fibrinogen, indicating an overall hypercoagulable tendency in this diabetic cohort. Diabetic Nephropathy was the most frequent complication (27.3%), followed by Diabetic Neuropathy (13.3%) and Diabetic Retinopathy (9.4%), with half the cohort having no complications (Table 1).

Table 2: Age and Sex Distribution Across Diagnostic Groups

Variable	Category	Nephropathy (n=35)	Neuropathy (n=17)	Retinopathy (n=12)	No Complications (n=64)	Total (n=128)
Age	31–40 yrs	0 (0.0%)	1 (5.9%)	0 (0.0%)	8 (12.5%)	9 (7.0%)
	41–50 yrs	7 (20.0%)	2 (11.8%)	3 (25.0%)	18 (28.1%)	30 (23.4%)
	51–60 yrs	18 (51.4%)	7 (41.2%)	6 (50.0%)	24 (37.5%)	55 (43.0%)
	≥61 yrs	10 (28.6%)	7 (41.2%)	3 (25.0%)	14 (21.9%)	34 (26.6%)
Sex	Female	16 (45.7%)	8 (47.1%)	4 (33.3%)	33 (51.6%)	61 (47.7%)
	Male	19 (54.3%)	9 (52.9%)	8 (66.7%)	31 (48.4%)	67 (52.3%)

Age: Pearson $\chi^2 = 10.976$, df = 9, p = 0.277 (NS) | Sex: Pearson $\chi^2 = 1.434$, df = 3, p = 0.698 (NS)

Age and sex were comparably distributed across all four diagnostic groups, with no statistically significant differences (p = 0.277 and p = 0.698 respectively), confirming that neither variable confounded the group comparisons (Table 2).

Table 3: Distribution of Coagulation Parameter Categories Across Diagnostic Groups

Parameter	Category	Nephropathy (n=35)	Neuropathy (n=17)	Retinopathy (n=12)	No Complications (n=64)	χ^2 (df), p-value
PT	≤11.9 s	15 (42.9%)	10 (58.8%)	5 (41.7%)	40 (62.5%)	6.481 (6), 0.372
	12.0–16.5 s	18 (51.4%)	6 (35.3%)	5 (41.7%)	21 (32.8%)	
	>16.5 s	2 (5.7%)	1 (5.9%)	2 (16.7%)	3 (4.7%)	
aPTT	≤26.9 s	17 (48.6%)	10 (58.8%)	5 (41.7%)	45 (70.3%)	8.056 (6), 0.234
	27.0–42.5 s	18 (51.4%)	7 (41.2%)	7 (58.3%)	18 (28.1%)	
	>42.5 s	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (1.6%)	
Fibrinogen	<2.0 g/L	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (1.6%)	104.182 (3), <0.001
	2.0–4.0 g/L	0 (0.0%)	0 (0.0%)	0 (0.0%)	58 (90.6%)	
	>4.0 g/L	35 (100%)	17 (100%)	12 (100%)	5 (7.8%)	

PT and aPTT category distributions did not differ significantly across the four groups (p = 0.372 and p = 0.234 respectively) (Table 3). In striking contrast, fibrinogen distribution differed significantly ($\chi^2 = 104.182$, p<0.001): every patient with

Diabetic Nephropathy, Neuropathy, or Retinopathy had elevated fibrinogen (>4.0 g/L), compared with only 7.8% of controls, of whom 90.6% had normal fibrinogen levels (Table 3).

Table 4: Comparison of Coagulation and Biochemical Parameters (Mean ± SD) Across Diagnostic Groups — One-Way ANOVA

Parameter	Nephropathy (n=35)	Neuropathy (n=17)	Retinopathy (n=12)	No Complications (n=64)	p-value
PT (s)	12.24 ± 2.45	11.87 ± 3.17	12.47 ± 3.22	11.70 ± 2.66	0.716
aPTT (s)	27.83 ± 5.43	26.76 ± 5.48	27.45 ± 4.96	26.28 ± 6.33	0.636
Fibrinogen (g/L)	5.06 ± 1.23	5.33 ± 1.03	5.18 ± 1.37	4.08 ± 1.14	<0.001
Bilirubin (mg/dL)	0.80 ± 0.48	0.73 ± 0.41	0.92 ± 0.59	0.92 ± 1.27	0.857
SGOT (U/L)	49.30 ± 39.42	38.28 ± 30.38	37.84 ± 23.71	62.34 ± 210.92	0.910
SGPT (U/L)	41.52 ± 34.13	35.16 ± 19.20	29.18 ± 13.53	74.57 ± 323.59	0.838
Total Protein (g/dL)	6.47 ± 1.70	6.31 ± 0.79	6.30 ± 1.20	6.57 ± 1.17	0.848
Albumin (g/dL)	3.20 ± 0.79	3.33 ± 0.79	3.30 ± 0.89	3.34 ± 0.83	0.866
S. Creatinine (mg/dL)	2.25 ± 1.78	1.56 ± 0.95	1.23 ± 0.43	2.30 ± 2.34	0.212
Blood Urea (mg/dL)	54.23 ± 35.48	42.23 ± 19.98	42.43 ± 15.18	51.22 ± 44.31	0.650
Total Cholesterol (mg/dL)	144.19 ± 52.90	141.85 ± 53.75	154.75 ± 54.41	148.30 ± 51.32	0.904
Triglycerides (mg/dL)	126.54 ± 87.31	93.87 ± 38.73	133.53 ± 77.10	147.04 ± 96.28	0.155
HbA1c (%)	8.25 ± 1.99	7.91 ± 0.74	9.07 ± 1.86	7.88 ± 1.49	0.115

Fibrinogen was the only parameter to differ significantly across groups ($F = 9.133$, $p<0.001$), with mean levels in all three complication groups exceeding the control group (4.08 ± 1.14 g/L). Mean PT and aPTT showed no significant inter-group difference ($p = 0.716$ and $p = 0.636$). None of the biochemical parameters — including HbA1c, serum creatinine, liver enzymes, or lipid profile — differed significantly across groups (all $p>0.05$), although mean HbA1c was numerically highest in Diabetic Retinopathy (9.07 ± 1.86%) and mean serum creatinine was highest in Diabetic Nephropathy (2.25 ± 1.78 mg/dL), consistent with clinical expectation despite not reaching statistical significance (Table 4). This confirms that the coagulation differences observed were not confounded by hepatic, renal, or metabolic abnormalities.

Discussion

Type II Diabetes Mellitus (T2DM) is a chronic metabolic disorder in which hyperglycaemia and insulin resistance upregulate procoagulant mediators — tissue factor and coagulation factors VII, IX, XI, and XII — while simultaneously impairing fibrinolysis through reduced tissue plasminogen activator and elevated plasminogen activator inhibitor-1, collectively producing a prothrombotic milieu now recognised as central to the pathogenesis of diabetic microvascular complications.[11] Studies systematically comparing PT, aPTT, and fibrinogen across specific complication subgroups,

particularly from Indian settings, remain limited, which formed the rationale for this study of 128 T2DM patients. In the present cohort, mean PT (11.94 ± 2.71 s) and aPTT (26.88 ± 5.84 s) were at the lower end of their reference ranges, with 54.7% and 60.2% of patients showing shortened values respectively — findings consistent with Saikia et al.,[11] Arun Kumar et al.,[12] and Oyakhire et al.,[13] who similarly reported shortened PT and aPTT in T2DM, particularly among patients with complications and poor glycaemic control. Kulkarni et al.[14] and Agarwal et al.[15] reported significantly shortened aPTT without a corresponding change in PT, partially concordant with the present findings. However, unlike these studies, the present cohort showed no statistically significant group-wise difference in PT or aPTT ($p=0.716$ and $p=0.636$), most plausibly because all 128 subjects were diabetic, precluding comparison against a non-diabetic control arm.

In contrast, fibrinogen emerged as the most discriminating coagulation parameter in this study, differing significantly across diagnostic groups ($F=9.133$, $p<0.001$); all patients with diabetic nephropathy, neuropathy, or retinopathy had fibrinogen levels exceeding 4.0 g/L, compared with only 7.8% of controls. This finding is strongly concordant with Mishra et al.,[16] Madan et al.,[17] Sherin et al.,[18] and Agarwal et al.,[15] who collectively documented significantly elevated fibrinogen in T2DM patients with microvascular complications and poor glycaemic control, attributable to hyperglycaemia-enhanced hepatic fibrinogen synthesis, fibrinogen glycation, and insulin resistance. Within the nephropathy subgroup, the universally elevated fibrinogen (5.06 ± 1.23 g/L) paralleled the findings of Mishra et al.[16] and Madan et al.,[17] who linked elevated fibrinogen and PAI-1 to glomerular microthrombosis and mesangial expansion, while the highest serum creatinine in this subgroup corroborated the expected pattern of renal impairment. In the neuropathy subgroup, the highest mean fibrinogen (5.33 ± 1.03 g/L) among all complication groups aligned with Mishra et al.,[16] supporting a role for endoneurial capillary ischaemia in nerve damage, although this contrasted with Madan et al.,[17] who found no significant haemostatic association with neuropathy — a discrepancy likely reflecting differences in the specific markers assessed and subgroup size. In the retinopathy subgroup, elevated fibrinogen (5.18 ± 1.37 g/L) alongside the highest mean HbA1c (9.07 ± 1.86%) was concordant with Oyakhire et al.,[13] though it diverged from Mishra et al.,[16] who reported paradoxically reduced fibrinogen in retinopathy, possibly reflecting local fibrinogen consumption within retinal microthrombi; Madan et al.[17] additionally implicated reduced Protein S and elevated von Willebrand factor as retinopathy-specific haemostatic markers not assessed in the present study.

Notably, none of the biochemical parameters assessed — including HbA1c, serum creatinine, liver function tests, and lipid profile — differed significantly across the four groups (all $p>0.05$), indicating that the observed fibrinogen elevation was independent of confounding hepatic, renal, or metabolic derangements. Taken together with the wider literature,[11–18] these findings confirm that among the standard coagulation screening parameters, fibrinogen alone consistently and robustly differentiates T2DM patients with microvascular complications from those without, regardless of the specific complication type, while PT and aPTT — though trending toward hypercoagulable (shortened) values — lack discriminatory power in an exclusively diabetic cohort. This supports incorporation of fibrinogen estimation into routine coagulation profiling of T2DM patients as a

simple, cost-effective adjunct for identifying individuals at higher risk of microvascular complications.

Conclusion

This cross-sectional study of 128 patients with Type II Diabetes Mellitus demonstrates that hyperfibrinogenaemia is strongly and significantly associated with the presence of microvascular complications, irrespective of whether the complication was nephropathy, neuropathy, or retinopathy. Fibrinogen was the only coagulation parameter that significantly differentiated T2DM patients with complications from those without ($p<0.001$), while PT and aPTT, despite trending toward shortened (hypercoagulable) values, did not differ significantly across diagnostic groups. The absence of significant differences in HbA1c, renal function, liver function, and lipid parameters across groups confirms that the fibrinogen elevation observed represents an independent marker of microvascular complication burden, rather than a consequence of confounding metabolic derangement. These findings support the role of a hypercoagulable state — particularly hyperfibrinogenaemia — as a mechanistic contributor to the pathogenesis of diabetic microvascular complications, and underscore the potential clinical value of incorporating routine fibrinogen estimation into the coagulation profiling of patients with Type II Diabetes Mellitus for early risk identification.

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