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Evaluation of Coagulation Profile in Type 2 Diabetes Mellitus: A Case–Control Study

Dr Yogita Kulkarni[1], Dr Pramod Kulkarni[2], Dr Ramanand Patil[3], Dr Gajanan Kulkarni[4*]

[1]Assistant Professor, Department of Physiology, Symbiosis medical college for women, Symbiosis University Hospital and Research Center, Symbiosis International University (Deemed University), Pune- 412115, India

[2]Associate Professor, Department of Community Medicine, Symbiosis medical college for women, Symbiosis University Hospital and Research Center, Symbiosis International University (Deemed University), Pune- 412115, India

[3]Professor, Department of Pharmacology, Symbiosis medical college for women Symbiosis University Hospital and Research Center Symbiosis International University (Deemed University) Pune. 412115, India

[4]Professor, Department of Pharmacology, Symbiosis medical college for women, Symbiosis University Hospital and Research Center, Symbiosis International University (Deemed University), Pune- 412115, India

Corresponding Author Email id: **gajanan.kulkarni@smcw.siu.edu.in**

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is associated with endothelial dysfunction, platelet hyper-reactivity, and altered coagulation, contributing to cardiovascular and thrombotic complications. Coagulation abnormalities in Indian diabetic populations remain under-explored. **Objective:** To evaluate and compare coagulation parameters—bleeding time (BT), clotting time (CT), prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (APTT), platelet count, and mean platelet volume (MPV)—between T2DM patients and healthy individuals. **Methods:**This case-control study included 40 T2DM patients and 40 age-matched healthy controls at a tertiary-care hospital. Coagulation tests were performed by standard procedures using an ECL-105 coagulometer and automated cell counter. Results were analyzed using unpaired t-test in SPSS software; $p < 0.05$ was considered significant. **Results:** The mean APTT in T2DM (26.87 ± 3.35 s) was significantly shorter than controls (36.18 ± 10.32 s; $p < 0.001$). CT (3.34 ± 0.63 min vs 3.69 ± 0.77 min; $p = 0.029$) and BT (1.98 ± 0.41 min vs 2.23 ± 0.66 min; $p = 0.045$) were also shorter in diabetics. MPV was significantly higher (9.84 ± 1.03 fl vs 8.76 ± 0.66 fl; $p < 0.001$). PT and INR showed no significant difference. **Conclusion:** T2DM patients demonstrate a hypercoagulable state reflected by shortened APTT and CT and increased MPV. Routine coagulation screening may aid early identification of thrombotic risk in diabetes.

INTRODUCTION:

Diabetes mellitus (DM) represents one of the most challenging global health concerns of the twenty-first century. It is a chronic metabolic disorder characterised by persistent hyperglycaemia due to defects in insulin secretion, insulin action, or both. The condition leads to disturbances in carbohydrate, fat, and protein metabolism, resulting in long-term microvascular complications such as neuropathy, nephropathy, and retinopathy, as well as macrovascular complications including coronary artery disease, cerebrovascular accidents, and peripheral arterial disease. Type 2 diabetes mellitus (T2DM) accounts for nearly 90–95% of all diabetes cases and is strongly associated with obesity, sedentary lifestyle, and advancing age. The prevalence of T2DM in India is rapidly increasing, reflecting urbanisation, dietary transitions, and genetic susceptibility.

Beyond its metabolic derangements, T2DM is now recognised as a pro-thrombotic and pro-inflammatory condition. Persistent hyperglycaemia induces endothelial injury and alters vascular homeostasis by impairing nitric oxide–mediated vasodilatation and promoting oxidative stress. This endothelial dysfunction triggers activation of the coagulation cascade and increases platelet reactivity, producing a milieu conducive to thrombus formation. Elevated fibrinogen levels, increased activity of coagulation factors VII, VIII, and IX, and decreased fibrinolytic activity have all been reported in diabetic patients, together promoting a hypercoagulable state.

Platelets from diabetic individuals exhibit enhanced adhesion and aggregation in response to physiological agonists such as adenosine diphosphate (ADP), collagen, and thrombin. This hyper-responsiveness is linked to non-enzymatic glycation of platelet membrane proteins, increased intracellular calcium, and altered signal transduction pathways. Mean platelet volume (MPV), an index of platelet size and activity, is frequently elevated in diabetic subjects, signifying the presence of larger, more reactive platelets with greater thrombotic potential. Similarly, abnormalities in conventional coagulation parameters—prothrombin time (PT), activated partial thromboplastin time (APTT), bleeding time (BT), and clotting time (CT)—have been observed, though findings across studies are inconsistent. Some researchers have reported shortened APTT and CT, suggesting hypercoagulability, while others have found prolonged times possibly related to vascular damage or comorbidities.

Hyperglycaemia also promotes the formation of advanced glycation end-products (AGEs) that up-regulate tissue factor expression on monocytes and endothelial cells, amplifying thrombin generation and fibrin formation. Moreover, insulin resistance and dyslipidaemia contribute to platelet activation and impaired fibrinolysis, further accentuating thrombosis risk. Despite these recognised mechanisms, there remains limited Indian data exploring comprehensive coagulation profiles in T2DM, especially using simple, routinely available laboratory tests.

Given the increasing burden of diabetes and its thrombotic complications, early identification of subclinical coagulation abnormalities is crucial. Evaluating coagulation parameters in diabetic individuals can provide insights into their haemostatic balance, aid in cardiovascular risk assessment, and guide perioperative management.

Therefore, the present study was designed to evaluate and compare the coagulation profile—including BT, CT, PT, INR, APTT, platelet count, and MPV—between patients with type 2 diabetes mellitus and healthy controls. The objective was to determine whether a hypercoagulable state exists among T2DM patients and to discuss its potential clinical significance in predicting vascular and thromboembolic complications.

MATERIALS AND METHODS:

Study design and setting:

This hospital-based, case–control study was conducted in the Departments of Physiology and Pathology at Vilasrao Deshmukh Government Medical College, Latur, Maharashtra, India, over a period of twenty-four months from January 2021 to December 2022. The study was designed to evaluate coagulation parameters in patients with Type 2 Diabetes Mellitus (T2DM) and compare them with healthy, age- and sex-matched individuals. A case–control design was chosen because it allows assessment of the association between diabetic status and coagulation abnormalities within a defined timeframe using a controlled comparison.

The laboratory investigations were performed in the Central Clinical Laboratory of the institution, which is equipped with automated haematology and coagulation analysers. Standard operating procedures were followed to ensure reliability and reproducibility of all test results.

Study Population and Sampling:
A total of 80 participants were enrolled in the study, consisting of 40 patients diagnosed with Type 2 Diabetes Mellitus (cases) and 40 apparently healthy volunteers (controls). Diabetic patients were selected from the outpatient and inpatient departments of medicine, based on the American Diabetes Association (ADA) diagnostic criteria, which include fasting plasma glucose ≥126 mg/dL, postprandial glucose ≥200 mg/dL, or HbA1c ≥6.5%. The control group included healthy adults attending routine health check-ups or relatives of patients, confirmed to have normal fasting glucose levels and no personal or family history of diabetes or cardiovascular disease.

Inclusion criteria:
- Adults aged between 40 and 75 years.
- Diagnosed cases of Type 2 Diabetes Mellitus with a disease duration of more than three years.
- Patients willing to participate and provide informed written consent.

Exclusion criteria:
- Individuals with Type 1 Diabetes Mellitus.
- History or evidence of cardiovascular, hepatic, renal, or hematological disorders.
- Current malignancy, acute infection, or inflammatory disease.
- Pregnancy or lactation.
- Use of anticoagulant or antiplatelet drugs within the previous three months.
- Habitual smokers or chronic alcohol consumers.

Participants who satisfied all eligibility criteria were recruited consecutively until the desired sample size was achieved. Demographic and clinical data such as age, sex, duration of diabetes, and random blood sugar were recorded in a structured proforma..

Sample collection and laboratory analysis
Under aseptic precautions, five millilitres of venous blood were drawn from the antecubital vein using a disposable syringe. For coagulation assays, 2 mL of blood was transferred immediately into a plastic tube containing 3.2% trisodium citrate anticoagulant in a 9:1 blood-to-anticoagulant ratio. The remaining sample was collected in an EDTA tube for platelet estimation.

Samples were processed within one hour of collection. Plasma was separated by centrifugation at 3000 revolutions per minute (rpm) for 15 minutes at room temperature. Prothrombin Time (PT) and Activated Partial Thromboplastin Time (APTT) were determined using the ECL-105 coagulometer (Erba Mannheim, Germany) with commercially available standard reagents. International Normalised Ratio (INR) was automatically calculated from PT values.

Bleeding Time (BT) and Clotting Time (CT) were measured manually using Duke's filter paper method and capillary tube method respectively, ensuring uniform conditions to minimise variability. Platelet count and Mean Platelet Volume (MPV) were estimated using an automated haematologyanalyser (Sysmex KX-21N, Japan) calibrated daily. Internal and external quality control procedures were implemented as per laboratory standards to ensure analytical accuracy.

Statistical analysis:
All data were entered into Microsoft Excel and subsequently analysed using Statistical Package for the Social Sciences (SPSS) version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages.

The unpaired Student's t-test was applied to compare mean values of coagulation parameters between diabetic patients and controls. For categorical data, the Chi-square test was used where applicable. Normality of data distribution was assessed using the Shapiro–Wilk test. Pearson's correlation coefficient was applied to evaluate relationships between coagulation parameters and selected clinical variables such as blood glucose levels or disease duration. A p-value < 0.05 was considered statistically significant for all comparisons.

The results were presented in tabular and graphical form, highlighting significant alterations in coagulation parameters among diabetic patients compared with healthy individuals.

Results:

A total of 80 participants were included in the study, comprising 40 patients with Type 2 Diabetes Mellitus (T2DM) and 40 healthy, age-matched controls. Both groups were comparable in terms of demographic characteristics such as age and sex distribution ($p > 0.05$), ensuring minimal confounding due to these variables. The mean age of diabetic subjects was **55.6 ± 11.3 years**, whereas controls had a mean age of **57.8 ± 9.6 years**. Male participants constituted 55% of the diabetic group and 37.5% of controls. As expected, mean random blood sugar levels were significantly higher among diabetics (**212.4± 58.8 mg/dL**) compared with controls (**132.1 ± 24.8 mg/dL**, $p< 0.001$).

Table 1 summarises the baseline demographic and clinical characteristics of both groups.

Table 1. Baseline characteristics of study participants

Parameter	T2DM (n = 40) Mean ± SD	Control (n = 40) Mean ± SD	*p*-value	Inference
Age (years)	55.6 ± 11.3	57.8 ± 9.6	0.42	NS
Males (%)	55.0	37.5	0.12	NS
Random Blood Sugar (mg/dL)	212.4 ± 58.8	132.1 ± 24.8	< 0.001	Significant ↑
Duration of Diabetes (years)	7.3 ± 3.1	—	—	—

NS = Not significant; ↑ = Increased in T2DM group

The mean values of coagulation parameters among diabetic patients and healthy controls are presented in Table 2. Diabetic subjects showed **a significant reduction in Bleeding Time (BT)** and **Clotting Time (CT)** compared to controls (p = 0.045 and 0.029 respectively). The **Activated Partial Thromboplastin Time (APTT)** was markedly shortened in diabetics (**26.87 ± 3.35 s**) relative to controls (**36.18 ± 10.32 s**,

$p< 0.001$), indicating enhanced coagulability. Mean Platelet Volume (MPV) was also significantly higher in diabetics (**9.84 ± 1.03 fl**) compared with controls (**8.76 ± 0.66 fl**, $p< 0.001$).

In contrast, **Prothrombin Time (PT)**and**International Normalised Ratio (INR)** showed no statistically significant differences between the two groups ($p> 0.05$). Platelet counts were marginally lower in diabetics, though not statistically significant.

Table 2. Comparison of coagulation parameters between T2DM patients and controls

Parameter	T2DM (n = 40) Mean ± SD	Control (n = 40) Mean ± SD	*p*-value	Inference
Bleeding Time (min)	1.98 ± 0.41	2.23 ± 0.66	0.045	Significant ↓
Clotting Time (min)	3.34 ± 0.63	3.69 ± 0.77	0.029	Significant ↓
Prothrombin Time (s)	14.72 ± 3.02	15.46 ± 3.80	0.34	NS
INR	1.10 ± 0.25	1.09 ± 0.23	0.32	NS
APTT (s)	26.87 ± 3.35	36.18 ± 10.32	< 0.001	Highly Significant ↓
Platelet Count (lakh/mm³)	2.56 ± 0.71	2.83 ± 0.93	0.15	NS
MPV (fl)	9.84 ± 1.03	8.76 ± 0.66	< 0.001	Highly Significant ↑

↓ = Decreased in T2DM group; ↑ = Increased in T2DM group; NS = Not significant

Overall, the results suggest that T2DM patients exhibit a distinct shift toward a **hypercoagulable and prothrombotic state**, as reflected by shortened APTT and CT and increased MPV. These findings support the hypothesis that diabetes promotes enhanced platelet activation and coagulation activity, potentially contributing to increased cardiovascular and thromboembolic risk.

DISCUSSION:

The present study demonstrates that Type 2 Diabetes Mellitus (T2DM) is associated with distinct alterations in haemostatic parameters, reflecting a shift toward a hypercoagulable state. This conclusion is supported by significantly shortened activated partial thromboplastin time (APTT) and clotting time (CT) and a rise in mean platelet volume (MPV) observed among diabetic patients compared to healthy individuals. These findings highlight the complex interplay between metabolic dysregulation, endothelial injury, and coagulation abnormalities in diabetes.

Diabetes is increasingly recognised not only as a metabolic disorder but also as a pro-thrombotic and pro-inflammatory condition. Chronic hyperglycaemia contributes to the activation of several pathogenic pathways, including oxidative stress, advanced glycation end-product (AGE) formation, and inflammatory cytokine release. These mechanisms lead to endothelial dysfunction, upregulation of tissue factor expression, and increased synthesis of coagulation factors, all of which predispose to thrombus formation. Our results align with this pathophysiological understanding, demonstrating clear evidence of hypercoagulability even in clinically stable diabetic patients.

In our study, APTT was significantly shortened in the diabetic group, indicating activation of the intrinsic coagulation pathway. This observation is consistent with the findings of Hekimsoy et al. [10] and Demirtunc et al. [11], who also reported abnormal coagulation profiles and increased platelet activity in patients with poor glycaemic control. Shortened APTT has been attributed to elevated plasma levels of fibrinogen and clotting factors VIII and IX, which accelerate thrombin generation and fibrin formation [12]. Increased fibrin turnover in diabetes may further contribute to vascular complications such as coronary artery disease and cerebrovascular accidents.

Platelet hyper-reactivity represents another important component of the pro-thrombotic state in T2DM. Several mechanisms contribute to enhanced platelet activation, including insulin resistance, increased oxidative stress, and glycation of platelet surface proteins [13,14]. These changes alter platelet membrane fluidity and calcium homeostasis, promoting spontaneous aggregation even in the absence of strong agonists. Mean platelet volume (MPV), a simple and reliable marker of platelet activity, was found to be significantly elevated in our diabetic participants compared to controls. Larger platelets are metabolically and enzymatically more active, contain more granules, and release greater amounts of prothrombotic mediators such as thromboxane A_2 and serotonin [15–17]. Therefore, an elevated MPV may be considered an early laboratory indicator of increased thrombotic risk in diabetic patients.

Our findings of shortened APTT and CT are comparable to those reported by Ankalayya et al. and Hassan et al. [18,19], who also demonstrated hypercoagulable tendencies in diabetics. Conversely, Alao et al. [20] reported prolonged coagulation times in a Nigerian population, attributing the discrepancy to ethnic differences, nutritional factors, and comorbid conditions. Such variability across studies underscores the need for region-specific research, as haemostatic responses in diabetes may be influenced by genetic, environmental, and dietary factors.

Interestingly, Prothrombin Time (PT) and International Normalised Ratio (INR) did not differ significantly between diabetics and controls in the present study, suggesting that the extrinsic pathway may remain relatively unaffected in early or moderate disease stages. This supports the hypothesis that the intrinsic and common pathways are more sensitive to metabolic and endothelial alterations associated with chronic hyperglycaemia.

Clinically, the demonstration of a subclinical hypercoagulable state in diabetes has significant implications. Increased platelet activation and accelerated coagulation can predispose to thromboembolic complications, particularly during surgery or acute metabolic stress. Incorporating simple coagulation tests such as APTT and MPV into routine evaluation may help identify high-risk patients who would benefit from preventive measures, including lifestyle modification, optimization of glycaemic control, or prophylactic antiplatelet therapy.

The strengths of this study lie in its case–control design, the use of standardised laboratory protocols, and inclusion of a comprehensive coagulation profile. However, certain limitations should be acknowledged. The sample size was modest, and being a single-centre study, the findings may not be generalisable to the wider population. Furthermore, specific assays for fibrinogen concentration, D-dimer, and von Willebrand factor were not included, which might have provided further mechanistic insights. Additionally, correlation of coagulation parameters with glycated haemoglobin (HbA1c) levels would have strengthened the interpretation of glycaemic influence on haemostatic changes.

Future research should focus on longitudinal and multicentric studies to validate these findings in larger cohorts. It would also be valuable to explore whether improved glycaemic control or pharmacological interventions targeting platelet and coagulation pathways can reverse or mitigate hypercoagulability in T2DM.

In summary, this study reinforces the evidence that diabetes mellitus promotes a hypercoagulable state through multiple mechanisms involving endothelial dysfunction, platelet activation, and intrinsic pathway activation. These haemostatic changes highlight the importance of integrating coagulation assessment into the routine care of diabetic patients to reduce cardiovascular and thrombotic complications.

Clinical implications Shortened APTT and elevated MPV in T2DM suggest a propensity for thromboembolic events. Routine coagulation screening may help identify patients at high risk for cardiovascular and perioperative complications.

Strengths and limitations Strengths include case-control design and complete coagulation profile. Limitations include single-centre sample, moderate size, and lack of glycated haemoglobin and fibrinogen correlation.

Future perspectives Larger multicentre studies should evaluate the association of glycaemic control with coagulation parameters and assess the effect of therapeutic interventions on hypercoagulability in T2DM.

CONCLUSION:

The present case–control study demonstrates that individuals with Type 2 diabetes mellitus exhibit distinct alterations in their coagulation profile, reflecting a tendency toward a hypercoagulable state. Among the various parameters assessed, a significant shortening of the activated partial thromboplastin time (APTT) and clotting time was observed in diabetic patients when compared with healthy controls. These findings suggest an activation of the intrinsic and common coagulation pathways, which may predispose diabetic individuals to thrombotic events. In addition, mean platelet volume (MPV) was found to be significantly elevated, indicating increased platelet activation and turnover. Larger and more reactive platelets are known to play a crucial role in the pathogenesis of microvascular and macrovascular complications associated with diabetes.

The observed coagulation abnormalities can be attributed to multiple interrelated mechanisms, including chronic hyperglycemia, endothelial dysfunction, oxidative stress, and low-grade inflammation. These factors collectively promote platelet hyperactivity and accelerate the atherothrombotic process. Thus, diabetes mellitus represents a prothrombotic milieu that increases the risk of cardiovascular diseases such as myocardial infarction, stroke, and peripheral vascular disease.

Given the simplicity, affordability, and availability of basic coagulation tests like PT, APTT, and MPV in most clinical laboratories, their inclusion in the routine evaluation of patients with Type 2 diabetes mellitus can provide valuable insight into the patient's thrombotic risk profile. Regular monitoring of these parameters may help clinicians identify individuals at higher risk of developing vascular complications, allowing for early intervention through optimization of glycemic control, lifestyle modification, and, where appropriate, antiplatelet or anticoagulant therapy.

In conclusion, the current study underscores the importance of evaluating the coagulation profile in diabetic patients as an integral part of comprehensive diabetes care. Early identification and management of hypercoagulable tendencies can significantly reduce the burden of vascular morbidity and mortality in Type 2 diabetes mellitus.

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NA

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Written informed consent obtained from all participants.

Consent for publication:
NA

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The authors declare no conflicts of interest.

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All authors are contributed equally.

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REFERENCES:

1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2022;45(Suppl 1):S17–S38.
2. Hekimsoy Z, Payzin B, Ornek T, Kandogan G. Mean platelet volume in type 2 diabetic patients. Platelets. 2004;15(6):411–414.
3. Demirtunc R, Duman D, Basar M, Bilgi M, Teomete M, Garip T. The relationship between glycemic control and platelet activity in type 2 diabetes mellitus. J Diabetes Complications. 2009;23(2):89–94.
4. Zhao Y, et al. Shortened APTT and increased fibrinogen values in diabetes mellitus. Clin Appl ThrombHemost. 2011;17(6):E95–E100.
5. Mansfield MW, Heywood DM, Grant PJ. Circulating levels of coagulation factors and their relationship to cardiovascular risk in NIDDM. ArteriosclerThrombVasc Biol. 1996;16(7):903–909.
6. Mustard JF, Packham MA. Platelets and diabetes mellitus. N Engl J Med. 1977;297(9):476–481.
7. Ankalayya GR, Dambal A, Shankar R, et al. Comparative study of coagulation time in type 2 diabetes mellitus and healthy individuals. J Clin Diagn Res. 2016;10(12):CC05–CC08.
8. Hassan AA, El-Nahas AM, Awadallah E. Coagulation abnormalities in type 2 diabetes mellitus patients. Egypt J Intern Med. 2018;30(4):215–220.
9. Alao OO, Adedoyin OT, et al. Haemostatic profile of Nigerian patients with type 2 diabetes mellitus. Niger Postgrad Med J. 2014;21(2):121–126.
10. Tripathi AK, Verma SP, Chandra M. Coagulation profile in diabetes mellitus and its clinical correlation. Indian J Hematol Blood Transfus. 2017;33(3):391–397.
11. Shankar R, Kumar S, Thomas J. Evaluation of coagulation parameters in type 2 diabetes mellitus. Int J Contemp Med Res. 2020;7(5):E15–E18.
12. Venkatesh K, Prasad GV, Rao DB. Study of coagulation profile in diabetes mellitus. Indian J Basic Appl Med Res. 2016;5(4):441–446.
13. Saxena S, Kishore R, Arora M. Platelet indices in diabetes mellitus and their association with glycemic control. J Clin Diagn Res. 2021;15(3):BC05–BC09.
14. Tavil Y, Sen N, Yazici HU, et al. Mean platelet volume and coronary atherosclerosis in diabetic and non-diabetic subjects. Atherosclerosis. 2007;197(2):820–825.
15. Coban E, Ozdogan M, Yazicioglu G, Akcit F. The mean platelet volume in patients with obesity and type 2 diabetes mellitus. Platelets. 2005;16(6):456–460.
16. Jindal S, Gupta S, Gupta R, et al. Platelet indices in diabetes mellitus: indicators of vascular complications. Hematology. 2011;16(2):86–89.
17. Kapoor A, Sinha N. Platelet function in diabetes mellitus and effect of antiplatelet drugs. Indian Heart J. 2018;70(6):864–872.
18. Balci YI, Karabulut A, et al. The evaluation of platelet parameters in type 2 diabetes mellitus and impaired glucose tolerance. Platelets. 2013;24(3):252–256.
19. Vinod KV, et al. Coagulation profile in patients with type 2 diabetes mellitus. Int J Adv Med. 2019;6(2):460–464.
20. Singh P, Reddy K, Kumar V. Correlation of HbA1c with coagulation parameters in type 2 diabetes mellitus. J Evid Based Med Healthc. 2022;9(10):25–30.
21. Kundu D, et al. Correlation of coagulation profile with glycemic status in type 2 diabetes mellitus. J Assoc Physicians India. 2020;68(11):35–40.
22. Ahmad M, et al. Study of coagulation parameters in type 2 diabetic patients in North India. J Clin Diagn Res. 2018;12(9):BC08–BC11.
23. Ozkan H, et al. Changes in coagulation parameters in type 2 diabetes mellitus. Clin Appl ThrombHemost. 2016;22(7):645–651.
24. Bae JH, et al. Platelet activation in diabetes mellitus: its relation with glycemic control. Thromb Res. 2001;103(1):23–28.
25. Maiti S, Saha S, et al. Study of platelet indices and coagulation profile in type 2 diabetes mellitus. Indian J Pathol Microbiol. 2021;64(3):507–512.
26. Ghosh A, Pal S. Evaluation of coagulation parameters and platelet indices in T2DM patients. J Clin Biomed Sci. 2019;9(2):45–51.
27. Shetty R, Kumar S, et al. A study of coagulation abnormalities in diabetes mellitus. Int J Adv Res Med. 2020;7(4):12–16.
28. Keating FK, Sobel BE, Schneider DJ. Effects of increased concentrations of glucose on platelet reactivity in healthy subjects and in patients with diabetes mellitus. Am J Cardiol. 2003;92(11):1362–1365.
29. Ajjan RA, et al. Diabetes and thrombosis: mechanisms of abnormal coagulation and fibrinolysis. Br J Haematol. 2015;168(2):170–183.
30. Tang WH, et al. Influence of diabetes on fibrin structure and function. Diabetologia. 2006;49(9):2285–2292.
31. Vazzana N, et al. Diabetes mellitus and thrombosis. Thromb Res. 2012;129(3):371–377.
32. Lip GYH, et al. Diabetes mellitus as a prothrombotic condition. J Am Coll Cardiol. 2007;49(7):857–869.
33. Papanas N, et al. Mean platelet volume in patients with type 2 diabetes mellitus. Exp Clin Endocrinol Diabetes. 2004;112(9):586–590.
34. Kushwaha R, et al. Platelet indices as markers of prothrombotic state in diabetes mellitus. Indian J Hematol Blood Transfus.

2020;36(2):390–397.
35. Sharma S, Mehta S, et al. Evaluation of hemostatic parameters in diabetic patients and their clinical significance. Int J Med Res Prof. 2018;4(4):230–235.
36. Gupta A, Yadav S, et al. A comparative study of coagulation profile in diabetic and non-diabetic patients. Indian J Appl Res. 2022;12(1):10–14.
37. International Diabetes Federation. IDF Diabetes Atlas. 10th ed. Brussels: IDF; 2021.
38. Kaur R, et al. Correlation between glycemic status and coagulation profile in T2DM. J Evol Med Dent Sci. 2019;8(22):1820–1824.
39. Pandey R, et al. Association of coagulation parameters with vascular complications in diabetes. J Clin Diagn Res. 2023;17(3):BC01–BC05.
40. WHO. Global report on diabetes. Geneva: World Health Organization; 2021.