Electronic Journal of General Medicine
2026, 23(2), em722
e-ISSN: 2516-3507
https://www.ejgm.co.uk/

MODESTUM

Original Article

OPEN ACCESS

Differences of coagulation and fibrinolysis profiles in controlled and uncontrolled T2DM patients

Edward Kurnia Setiawan Limijadi [1*], Dwi Retnoningrum [1], Banundari Rachmawati [1], Nyoman Suci Widyastiti [1], Nur Farhanah [2], Anugrah Riansari [2], Danendra Rakha Putra Respati [3], Kevin Christian Tjandra [3], Fitri Mutmainnah [4], Meita Hendrianingtyas [1]

[1] Department of Clinical Pathology, Faculty of Medicine, Universitas Diponegoro, Semarang, INDONESIA
[2] Department of Internal Medicine, Faculty of Medicine, Universitas Diponegoro, Semarang, INDONESIA
[3] Faculty of Medicine, Universitas Diponegoro, Semarang, INDONESIA
[4] Faculty of Medicine, Universitas Andalas, Padang, INDONESIA
***Corresponding Author:** edwardksl@fk.undip.ac.id

Citation: Limijadi EKS, Retnoningrum D, Rachmawati B, Widyastiti NS, Farhanah N, Riansari A, Respati DRP, Tjandra KC, Mutmainnah F, Hendrianingtyas M. Differences of coagulation and fibrinolysis profiles in controlled and uncontrolled T2DM patients. Electron J Gen Med. 2026;23(2):em722. https://doi.org/10.29333/ejgm/18087

ARTICLE INFO

Received: 14 May 2025

Accepted: 18 Feb. 2026

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is one of the degenerative diseases that continues to increase. Diabetes is one of the three biggest causes of death in Indonesia in 2017. This burden is exacerbated by the presence of uncontrolled diabetes cases, which have a negative impact on almost every system of the human body. Coagulopathy, which is defined as a disorder of the blood clotting process, is one of the clinical manifestations of uncontrolled T2DM. Chronic hyperglycemia in T2DM can affect the hypercoagulation process which includes changes in platelet activation time, endothelial cell function, and fibrinolysis.

Objective: To determine and analyze the differences in coagulation and fibrinolysis profiles of controlled and uncontrolled T2DM patients.

Method: The design of this study was cross-sectional with a target population of adult T2DM patients at the Diponegoro National Hospital in Semarang City. Data collection on respondents includes filling out questionnaires and taking blood samples to examine the coagulation and fibrinolysis profiles and Hba1C levels of respondents

Results: There were no abnormalities in prothrombin time (PT) and activated partial prothrombin time (APTT) values in the two groups of this study and there was also no difference in the mean values of PT and APTT between the two groups in this study. Increased D-dimer concentrations occurred in both groups but there was no significant difference between the two groups ($p > 0.05$).

Conclusion: There was no significant difference in PT, APTT, and D-dimer values in the two groups of the study.

Keywords: type 2 diabetes mellitus, activated partial prothrombin time, prothrombin time

INTRODUCTION

According to International Diabetes Federation in 2021, global prevalence of type 2 diabetes mellitus (T2DM) is 536.6 million cases (10.5%) in adults and will continue to increase to 783.2 cases (12.2%) of T2DM worldwide in 2045 [1]. Indonesia is one of the developing countries with the most T2DM case in the world. In 2019, it was reported that there were 10.9 million case in Indonesia and this number is expected to continue to increase to reach 16.6 million in 2045 [2]. Semarang City Health Office revealed that throughout 2023, T2DM cases in Semarang City had reached 5,991 cases, which was an increase from the previous year [3].

Diabetes mellitus (DM) is one of biggest causes of death in Indonesia based on Basic Health Research 2017 [4]. This burden is exacerbated by the presence of uncontrolled diabetes cases that can trigger disorders of the hemostasis system [5, 6]. Hemostasis is body mechanism to stop bleeding spontaneously. Hemostasis aims to maintain blood thinness and close damage to blood vessel walls, thereby reducing blood loss when damage occurs. Vascular system, platelets, coagulation and fibrinolysis are related to each other in a homeostasis system. Hyperglycemia in T2DM not only disrupts metabolic balance but also contributes to endothelial dysfunction and inflammatory cytokine production, further exacerbating the prothrombotic state [7].

Previous studies have shown that female gender, older age, duration of T2DM, obesity, and patients with poor glycemic control can also increase the risk of coagulation disorder [8]. Hypercoagulable condition that occurs in patients with T2DM can trigger macroangiopathy and cardiovascular dysfunction. DM patients with HbA1c ≤ 7% tend to have a lower risk of recurrent angina. Chronic hyperglycemia in T2DM can affect the hypercoagulation process which includes changes in platelet activation time, endothelial cell function, and fibrinolysis [9]. This hypercoagulation is caused by an imbalance between the blood vessel endothelium and blood

coagulation factors [8]. The inflammatory process that also increases in T2DM patients is also suspected to be one of the causes of hypercoagulation in patients [10].

D-dimer is a soluble fibrin degradation product derived from the degradation of cross-linked fibrin mediated by plasmin. Therefore, D-dimer can be considered as a biomarker of coagulation and fibrinolysis activation [11]. Increased D-dimer indicates an increase in systemic fibrin formation and an increase in thrombosis production [12]. Activated partial prothrombin time (APTT) and prothrombin time (PT) are also blood test parameters that can be used to evaluate the patient's coagulation status. Shorter APTT and PTT times indicate an increase in thromboembolism formation that can trigger atherosclerosis in T2DM patients [13, 14]. Moreover, persistent hyperglycemia may lead to non-enzymatic glycation of coagulation proteins, altering their structure and function [15]. Recent studies also link oxidative stress and dyslipidemia in T2DM as contributors to the prothrombotic profile observed in affected individuals [16].

This study will analyze the differences in coagulation and fibrinolysis profiles of controlled and uncontrolled T2DM patients based on their Hba1C levels. This study is expected to be an additional reference for knowledge related to the analysis of T2DM with hypercoagulation events. Not many studies have been conducted on this topic. This study is expected to add reference materials to studies related to the topic as a scientific reference for compiling promotive and preventive efforts to prevent clinical manifestations that worsen the condition of T2DM patients.

METHOD

This study employed a cross-sectional design and was conducted from August to September 2024 at Diponegoro National Hospital, Semarang. The sampling technique used was purposive sampling according to the inclusion criteria. Participants were included based on the following criteria: male or female patients aged between 30 to 60 years, not experiencing fever at the time of examination, not taking anticoagulant medications, and willing to participate throughout the study by signing informed consent. Exclusion criteria included patients with a history or diagnosis of pulmonary disease, cardiovascular disease, liver disease, autoimmune disease, or kidney disease other than diabetic nephropathy. This purposive sampling approach has been recommended in clinical studies where strict inclusion criteria are necessary to minimize confounding variables [17].

A total of 60 patients with T2DM participated in this study. They were divided into two equal groups: the uncontrolled DM group, consisting of 30 patients with HbA1c levels above 7%, and the controlled DM group, consisting of 30 patients with HbA1c levels less than or equal to 7%. Ethical approval was obtained from the Research and Health Ethics Commission of the Faculty of Medicine, Diponegoro University with certificate number 257/ECC/KEPK/FK-UNDIP/V/2024.

Blood samples were collected from the median cubital vein, with a total of 6 cc of blood drawn from each participant. The blood was divided equally into two tubes, with 3 cc placed into a 3.6% sodium citrate tube, which was inverted eight times to ensure proper mixing for coagulation testing. This process could be conducted simultaneously with EDTA blood collection for hematological or other examinations. The samples were then centrifuged at 1,500 g for 10 minutes to obtain citrate plasma. Laboratory assessments included measurements of HbA1c, PT, APTT, and D-dimer levels.

Table 1. Characteristics of the respondents

Variable	HbA1C > 7	HbA1C ≤ 7	p
Age (years)	51.13 ± 11.25	52.09 ± 11.31	0.87
Male	12 (40 %)	12 (40 %)	
Female	18 (60%)	18 (60 %)	
Weight (kg)	72.43 ± 19.80	72.42 ± 19.55	0.98
BMI (kg/m^2)	29.08 ± 7.19	34.23 ± 40.84	0.94

PT, APTT, and D-dimer levels were measured using immunoturbidimetric and clot-based coagulation assays on the Coatron A4 analyzer (TECO GmbH, Germany). D-dimer quantification was performed using the blue D-dimer LC Kit (Cat. No. D2020-010), an immunoturbidimetric assay that utilizes latex particles coated with monoclonal antibodies, measured at 400-470 nm. For PT, the TEClot PT-S reagent (Cat. No. A0230-010), which contains rabbit brain extract and calcium chloride, was employed to assess the extrinsic coagulation pathway. APTT was measured using the TEClot APTT-S reagent (Cat. No. A0300-050), which includes phospholipids and colloidal silica as activators for evaluating the intrinsic pathway. All reagents were prepared and used according to the manufacturer's protocols, with strict adherence to temperature and handling guidelines to ensure assay accuracy and precision.

All data were analyzed using SPSS version 22. The normality of the data distribution was tested using the Kolmogorov-Smirnov test. Continuous variables with a normal distribution were presented as mean (M) ± standard deviation (SD), whereas those with a non-normal distribution were presented as median (minimum-maximum). Independent t-tests were used to compare normally distributed variables between the controlled and uncontrolled DM groups, while the Mann-Whitney U test was used for non-normally distributed variables. The relationship between variables was analyzed using Pearson or Spearman correlation tests, depending on the data distribution.

RESULTS

It can be seen **Table 1** that the comparison of respondents in the two groups in this study has the same number, namely 18 female respondents and 12 male respondents. The mean age of participants in the uncontrolled group was 51.13 ± 11.25 years, while in the controlled group it was 52.09 ± 11.31 years, showing no statistically significant difference between the groups (p = 0.87). Gender distribution was identical in both groups, with 12 males (40%) and 18 females (60%) in each group. The average body weight was similar between the groups, with the uncontrolled group averaging 72.43 ± 19.80 kg and the controlled group averaging 72.42 ± 19.55 kg (p = 0.98). Body mass index (BMI) also did not differ significantly between the two groups, with a mean of 29.08 ± 7.19 kg/m^2 in the uncontrolled group and 34.23 ± 40.84 kg/m^2 in the controlled group (p = 0.94).

Table 2 presents the baseline characteristics of hematological assessments in patients with uncontrolled and controlled T2DM. The hemoglobin levels were comparable between the two groups, with a mean of 13.44 ± 2.81 g/dL in the uncontrolled group and 13.31 ± 2.84 g/dL in the controlled

Table 2. Baseline characteristic of hematological assessment

Variable	HbA1C > 7	HbA1C ≤ 7	p
Hemoglobin (g/dl)	13.44 ± 2.81	13.31 ± 2.84	0.286
Leukocyte (10^3/uL)	8.11 ± 2.19	7.54 ± 2.38	0.226
Trombocyte (10^3/uL)	298.27 ± 76.90	263.33 ± 59.02	0.032*
FBS (mg/dl)	214.43 ± 94.78	114.45 ± 32.80	0.000*
HbA1c (%)	10.81 ± 1.87	5.67 ± 1.00	0.000*
Creatinin (mg/dl)	0.81 ± 1.40	0.86 ± 0.32	0.342
PT (second)	11.91 ± 2.81	12.16 ± 0.84	0.126
aPTT (second)	27.79 ± 4.36	28.72 ± 3.13	0.089
D-dimer (ng/mL)	208.63 ± 346.73	224.79 ± 263.57	0.454

Note. *p < 0.05 (Significant)

Table 3. Correlation between coagulation and fibrinolysis profiles with HbA1C

Characteristic	r-value	p
PT (second)	-0.097	0.462
APTT (second)	-0.092	0.484
D-dimer (ng/mL)	-0.358	0.005

group (p = 0.286). Leukocyte counts were also similar, with no statistically significant difference observed between the uncontrolled group (8.11 ± 2.19 × 10^3/uL) and the controlled group (7.54 ± 2.38 × 10^3/uL) (p = 0.226). Laboratory results such as leukocyte and platelet counts are valuable in assessing subclinical inflammation and vascular stress in diabetic patients [18].

A significant difference was found in platelet (thrombocyte) counts, where the uncontrolled DM group had a higher mean value (298.27 ± 76.90 × 10^3/uL) compared to the controlled group (263.33 ± 59.02 × 10^3/uL), with a p-value of 0.032. Fasting blood glucose (FBS) and HbA1c levels showed highly significant differences between the two groups. The uncontrolled group had substantially higher FBS (214.43 ± 94.78 mg/dL) and HbA1c levels (10.81 ± 1.87%) than the controlled group (114.45 ± 32.80 mg/dL and 5.67 ± 1.00%, respectively), both with p-values of 0.000. Higher thrombocyte counts in uncontrolled T2DM patients may reflect a compensatory response to endothelial injury [19].

Serum creatinine levels showed no significant difference between the two groups (0.81 ± 1.40 mg/dL vs. 0.86 ± 0.32 mg/dL; p = 0.342). Similarly, coagulation parameters such as PT and APTT did not differ significantly between the uncontrolled and controlled groups, with PT values of 11.91 ± 2.81 seconds and 12.16 ± 0.84 seconds (p = 0.126), and APTT values of 27.79 ± 4.36 seconds and 28.72 ± 3.13 seconds (p = 0.089), respectively. D-dimer levels were also comparable, with no statistically significant difference noted (208.63 ± 346.73 ng/mL in the uncontrolled group vs. 224.79 ± 263.57 ng/mL in the controlled group; p = 0.454).

Overall, significant differences were only observed in thrombocyte counts, FBS, and HbA1c levels, indicating that poor glycemic control may be associated with higher platelet counts and elevated glucose parameters. Other hematological and coagulation parameters did not show notable variations between the groups.

Table 3 presents the correlation between coagulation and fibrinolysis parameters with HbA1C levels. The results show a weak negative correlation between PT and HbA1C (r = -0.097, p = 0.462), as well as between APTT and HbA1C (r = -0.092, p = 0.484); however, both correlations are not statistically significant. In contrast, D-dimer levels exhibit a moderate negative correlation with HbA1C (r = -0.358), which is statistically significant (p = 0.005). This suggests that higher HbA1C levels may be associated with lower D-dimer concentrations, indicating a potential link between glycemic control and fibrinolytic activity.

DISCUSSION

Total respondents in this study were 60 respondents divided into 2 groups with each group consisting of 30 respondents based on HbA1C levels. Gender composition respondent in this study was also the same between the two groups, consisting of 40% Male and the remaining 60% Female. In accordance with previous studies which showed that women have a higher risk of suffering from T2DM when compared to men. Women tend to be more prone to obesity and psychological stress which are triggers for T2DM [20]. The average age of respondents in the study also did not show a significant difference between the two research groups, namely 51 years for the uncontrolled DM group and 52 for controlled DM.

Risk of developing T2DM will increase with age, especially over the age of 40 years and over [21]. Respondents in this study were T2DM sufferers, both controlled and uncontrolled, without complications of kidney disease, autoimmune disease, or other malignant diseases. Results of the blood analysis of respondents in this study were that the research respondents were in a healthy condition, indicated by the average hemoglobin levels of respondents in both the controlled and uncontrolled DM groups showing results of 13.44 for the uncontrolled DM group and 13.41 for controlled DM. The number of leukocytes and platelets also showed a normal average number between the two groups as well as for the results of creatinine (**Table 1**)

PT is a blood laboratory test to see how fast the blood clotting process occurs in each individual. One of the clinical disorders experienced by T2DM patients is a blood clotting disorder, one of the parameters of which is PT measurement. PT measurement is used to see extrinsic factors in the blood clotting process. Hyperglycemia conditions will trigger a decrease/shortening of PT because it is triggered by intracellular and extracellular protein glycation which can later change the normal function of these blood clotting factors, thereby affecting their functional capacity in the blood clotting process. The results of the analysis in this study showed that all respondents had PT values that were still within normal limits, namely between 11-15 seconds. Liver function plays a crucial role in PT regulation, and preserved liver status in early-stage T2DM patients may explain the normal PT findings [22]. There was no significant difference in either the group with higher HbA1C and the group of respondents who had HbA1C values less than or equal to 7. The results of this study are in accordance with several previous studies. The study showed that there was no significant difference in PT values between the healthy respondent group and respondents with T2DM [23]. Other studies have shown that PT in patients with DM tends to be shorter than the control group or people without DM [24]. The significant results in this study are likely due to the condition of the respondents in this study, most of whom did not experience other complications other than T2DM. The presence of complications caused by prolonged hyperglycemia, especially liver disorders, can cause PT abnormalities [25, 26].

APTT is a laboratory test used to assess the activity of intrinsic coagulation factors in the blood. Similar to PT, in this study there was no significant difference between the controlled and uncontrolled DM groups ($p > 0.05$). Most respondents in this study had APTT values that were still within normal limits. In previous studies, it was found that significant differences in APTT values were found in comparisons of diabetic patients with non-diabetic patients, while differences in the duration of diabetes did not cause significant differences in APTT values [27]. This insignificant difference indicates that respondents in this study had not experienced coagulation disorders because most respondents did not have comorbid diseases that could increase the risk of APTT value abnormalities. Interestingly, the duration of DM itself has not been shown to significantly influence APTT values in several studies [27]. The study in [24] reported that APTT values in T2DM patients were significantly lower than in healthy controls. APTT sensitivity to heparin and intrinsic pathway abnormalities makes it an essential but sometimes variable marker in diabetes-related coagulopathy [28].

Results of D-dimer analysis in both groups of respondents in this study showed that there was an increase in D-dimer in both groups of respondents, however, there was no significant difference in the mean of D-dimer in the two groups ($p > 0.05$). D-dimer in the uncontrolled and controlled DM groups were, respectively (208.63 ± 346.73) ng/mL and 224.79 ± 263.57 ng/mL. Results of this study are in line with previous studies that in the condition of DM in patients can increase the concentration of D-dimer in blood serum [12, 29]. Elevated D-dimer levels in early-stage T2DM may suggest the presence of silent endothelial dysfunction, even in the absence of clinical complications [30]. This fibrinogen elevation leads to the formation of denser, more resistant fibrin clots, which are harder to degrade by plasmin [31]. The absence of this significant difference is also likely due to the respondents in this study still being classified as early stage DM without complications even though they had high HbA1C levels [29].

In our cohort of 60 adults with T2DM (30 controlled vs. 30 uncontrolled by HbA1c), the two groups were comparable in baseline characteristics, with identical sex distribution (40% men; 60% women) and similar mean age (51 vs. 52 years). This pattern is clinically plausible because women with T2DM often present with a higher cardiometabolic risk-factor burden (particularly excess adiposity) and may be more affected by psychosocial stress pathways that contribute to dysglycemia and cardiometabolic risk [32]. Importantly, we limited inclusion to patients without major renal, autoimmune, malignant, or other systemic comorbidities, and routine hematologic/biochemical indices (hemoglobin, leukocytes, platelets, and creatinine) were within expected ranges across groups—minimizing confounding from overt inflammation, anemia, thrombocytopenia, or advanced organ dysfunction that could otherwise distort coagulation assays.

Regarding coagulation and fibrinolysis profiles, the absence of significant between-group differences in PT and APTT—together with values largely within reference ranges—should be interpreted in the context of contemporary evidence that global clotting times are relatively insensitive to the prothrombotic phenotype of T2DM, particularly in uncomplicated/early-stage disease. A recent systematic review and meta-analysis (Frontiers in Medicine, 2024) found that PT/APTT differences are more consistently observed when comparing diabetic patients with non-diabetic controls, whereas within-diabetes contrasts can be small and heterogeneous, and are influenced by comorbidity burden and study setting [33]. Mechanistically, modern reviews emphasize that diabetes-associated thrombogenicity is driven less by prolongation/shortening of screening clotting times and more by “qualitative” changes—enhanced thrombin generation, formation of denser fibrin networks, and hypofibrinolysis mediated by elevated PAI-1 and post-translational modifications (including glycation/oxidation) of fibrinogen and related proteins—yielding clots that are harder to lyse despite normal PT/APTT [34-36]. In this framework, the observed elevation of D-dimer in both groups, without a clear HbA1c-stratified difference, is consistent with low-grade, subclinical activation of coagulation/fibrin turnover that can occur even before overt vascular complications; clinically, higher D-dimer has been associated with increased cardiovascular event risk in T2DM populations, supporting its potential value as a risk-stratification marker rather than a direct discriminator of glycemic control alone [12]. Additionally, platelet-fibrinolysis interactions (including platelet-associated PAI-1 dynamics) may contribute to a hypofibrinolytic milieu even in “well-controlled” T2DM, which could partly explain why D-dimer elevations may persist across HbA1c strata in otherwise stable patients [37].

CONCLUSION

In this study, there is no abnormalities in PT and APTT values in patients with controlled or uncontrolled DM. There was an increase in D-dimer concentration so that it exceeded the recommended value for respondents in this study.

Author contributions: EKSL: conceptualization, methodology, writing – original draft, project administration; **DR:** conceptualization, validation, resources, writing – review & editing, supervision; **BR:** methodology, writing – review & editing; **NSW:** validation; **NF:** investigation, data curation; **AR:** formal analysis, investigation; **DRPR:** software, data curation, visualization; **KCT:** conceptualization, software, formal analysis, writing – original draft, visualization; **FM:** investigation; **MH:** methodology, resources, writing – review & editing, supervision, funding acquisition. All authors agreed with the results and conclusions.

Funding: No funding source is reported for this study.

Acknowledgments: The authors would like to thank the patients and their families for their kindness and willingness to participate.

Ethical statement: The authors stated that the study was approved by the Bioethics Committee at Universitas Diponegoro on 15 May 2018 with approval number RNN/166/18/KE. Written informed consents were obtained from the participants.

AI statement: The authors stated that, during the preparation of this manuscript, generative artificial intelligence (AI) tools were utilized to assist with language refinement, grammar correction, and improvement of sentence clarity. The AI tools were not used for data analysis, interpretation of results, generation of scientific content, or drawing conclusions. All intellectual content, study design, data collection, analysis, and interpretation were conducted independently by the authors. The authors have carefully reviewed and verified all outputs generated with AI assistance and take full responsibility for the content of this manuscript.

Declaration of interest: No conflict of interest is declared by the authors.

Data sharing statement: Data supporting the findings and conclusions are available upon request from the corresponding author.

REFERENCES

1. Sun H, Saeedi P, Karuranga S, et al. IDF Diabetes atlas: Global, regional and country-level diabetes prevalence estimates for 2021 and projections for 2045. Diabetes Res Clin Pract. 2022;183:109119. https://doi.org/10.1016/j.diabres.2021.109119 PMid:34879977 PMCid:PMC11057359
2. Hidayat B, Ramadani RV, Rudijanto A, Soewondo P, Suastika K, Siu Ng JY. Direct medical cost of type 2 diabetes mellitus and its associated complications in Indonesia. Value Health Reg Issues. 2022;28:82-9. https://doi.org/10.1016/j.vhri.2021.04.006 PMid:34839111
3. Dinas Kesehatan Kota Semmarang. Semarang City health profile. Institute for Health Metrics and Evaluation; 2018. Available at: http://www.healthdata.org/indonesia (Accessed: 11 March 2020).
4. Glasheen WP, Renda A, Dong Y. Diabetes complications severity index (DCSI)-update and ICD-10 translation. J Diabetes Complications. 2017;31(6):1007-13. https://doi.org/10.1016/j.jdiacomp.2017.02.018 PMid:28416120
5. Gomes MB, Rathmann W, Charbonnel B, et al. Treatment of type 2 diabetes mellitus worldwide: Baseline patient characteristics in the global DISCOVER study. Diabetes Res Clin Pract. 2019;151:20-32. https://doi.org/10.1016/j.diabres.2019.03.024 PMid:30904743
6. Sobczak AIS, Stewart AJ. Coagulatory defects in type-1 and type-2 diabetes. Int J Mol Sci. 2019;20(24):6345. https://doi.org/10.3390/ijms20246345 PMid:31888259 PMCid:PMC6940903
7. Carr ME. Diabetes mellitus: A hypercoagulable state. J Diabet Complicat. 2001;15(1):44-54. https://doi.org/10.1016/S1056-8727(00)00132-X PMid:11259926 PMCid:PMC7968529
8. Mwambungu A, Kaile T, Korolova L, Kwenda J, Marimo C. Risk factors associated with hypercoagulability in type 2 diabetes mellitus patients at Ndola Central Hospital Zambia. Med J Zambia. 2014;41(2):70-80.
9. Getu F, Aynalem M, Bizuneh S, Enawgaw B. The prevalence of coagulopathy and associated factors among adult type II diabetes mellitus patients attending the University of Gondar Comprehensive Specialized Hospital, Northwest Ethiopia. Diabetes Metab Syndr Obes. 2022;15:579-90. https://doi.org/10.2147/DMSO.S349412 PMid:35237057 PMCid:PMC8885125
10. Randeria SN, Thomson GJA, Nell TA, Roberts T, Pretorius E. Inflammatory cytokines in type 2 diabetes mellitus as facilitators of hypercoagulation and abnormal clot formation. Cardiovasc Diabetol. 2019;18(1):72. https://doi.org/10.1186/s12933-019-0870-9 PMid:31164120 PMCid:PMC6549308
11. Bembde AS. A study of plasma fibrinogen level in type-2 diabetes mellitus and its relation to glycemic control. Indian J Hematol Blood Transfus. 2012;28(2):105-8. https://doi.org/10.1007/s12288-011-0116-9 PMid:23730017 PMCid:PMC3332265
12. Cheng L, Fu Q, Zhou L, et al. D-dimer as a predictor of cardiovascular outcomes in patients with diabetes mellitus. BMC Cardiovasc Disord. 2022;22(1):82. https://doi.org/10.1186/s12872-022-02531-x PMid:35246061 PMCid:PMC8895572
13. Husanah FH, Siga AF, Sari SD. Korelasi HbA1c, APTT dan PT pada pasien diabetes militus tipe 2 sebagai prediktor hiperkoagulasi di rumah Sakit Umum Gambiran Kota Kediri [Correlation of HbA1c, APTT and PT in type 2 diabetes mellitus patients as a predictor of hypercoagulation at Gambiran General Hospital, Kediri City]. J Sintesis. 2021;2(2):97-104. https://doi.org/10.56399/jst.v2i2.24
14. Pabinger I, Thaler J, Ay C. Biomarkers for prediction of venous thromboembolism in cancer. Blood. 2013;122(12):2011-8. https://doi.org/10.1182/blood-2013-04-460147 PMid:23908470
15. Selvin E, Steffes MW, Zhu H, et al. Glycated hemoglobin, diabetes, and cardiovascular risk in nondiabetic adults. N Engl J Med. 2010;362:800-11. https://doi.org/10.1056/NEJMoa0908359 PMid:20200384 PMCid:PMC2872990
16. Zhuang L, Yu C, Xu F, et al. Increased plasma D-dimer levels may be a promising indicator for diabetic peripheral neuropathy in type 2 diabetes. Front Endocrinol. 2022;13:930271. https://doi.org/10.3389/fendo.2022.930271 PMid:36082076 PMCid:PMC9445160
17. Goyal R, Jialal I. Type 2 diabetes mellitus. Treasure Island (FL): StatPearls Publishing; 2020.
18. Madan R, Gupta B, Saluja S, Kansra UC, Tripathi BK, Guliani BP. Coagulation profile in diabetes mellitus and its association with microvascular complications. J Assoc Physicians India. 2010;58:481-4.
19. Randeria SN, Thomson GJA, Nell TA, Roberts T, Pretorius E. Inflammatory cytokines in type 2 diabetes mellitus as facilitators of hypercoagulation. Cardiovasc Diabetol. 2019;18(1):72. https://doi.org/10.1186/s12933-019-0870-9 PMid:31164120 PMCid:PMC6549308
20. Kautzky-Willer A, Leutner M, Harreiter J. Correction to: Sex differences in type 2 diabetes. Diabetologia. 2023;66(6):1165. https://doi.org/10.1007/s00125-023-05913-8
21. Yan Z, Cai M, Han X, Chen Q, Lu H. The interaction between age and risk factors for diabetes and prediabetes: A community-based cross-sectional study. Diabetes Metab Syndr Obes. 2023;16:85-93. https://doi.org/10.2147/DMSO.S390857 PMid:36760587 PMCid:PMC9843502
22. Bos MHA, van't Veer C, Reitsma PH. Molecular biology and biochemistry of the coagulation factors. In: Kaushansky K, Levi MM, eds. Williams hematology hemostasis and thrombosis. McGraw Hill; 2018.
23. Oyakhire FO, Adejumo BIG, Aiyegbusi OV, et al. Coagulation profile in diabetes mellitus and its association with microvascular complications in uncontrolled and controlled diabetes at Edo Specialist Hospital, Benin-City, South-South, Nigeria. Med Lab Technol J. 2023;9(1). https://doi.org/10.31964/mltj.v8i2.493
24. Thukral S, Hussain S, Bhat S, Kaur N, Reddy A. Prothrombin time (PT) and activated partial thromboplastin time (APTT) in type 2 diabetes mellitus, a case control study. Int J Contemp Med Res. 2018;5(8):5-9. https://doi.org/10.21276/ijcmr.2018.5.8.13
25. Ambelu YA, Shiferaw MB, Abebe M, Enawgaw B. Prothrombin time, activated partial thromboplastin time and platelet counts of type II diabetes mellitus: A comparative study. J Diabetes Metab Disord. 2018;17(2):117-21. https://doi.org/10.1007/s40200-018-0347-5 PMid:30918844 PMCid:PMC6405409

26. Mohammed OIA. Estimation of prothrombin time, activated partial thromboplastin time and thrombocytes among Sudanese patients with diabetes. GSC Biol Pharm Sci. 2020;10(1):34-8. https://doi.org/10.30574/gscbps.2020.10.1.0250
27. Sapkota B, Shrestha SK, Poudel S. Association of activated partial thromboplastin time and fibrinogen level in patients with type II diabetes mellitus. BMC Res Notes. 2013;6:485. https://doi.org/10.1186/1756-0500-6-485 PMid:24274772 PMCid:PMC4222085
28. Fritsma MG, Fritsma GA. Normal hemostasis and coagulation. In: Keohane EM, Smith LJ, Walenga JM, eds. Rodak's hematology clinical principles and applications. 5th ed. Missouri: Elsevier; 2016. p. 642-66.
29. Kanani D, Vaidehipatel, Kiranchauhan, Haridas N. Association of D-dimer in type 2 diabetes mellitus. Int J Adv Res. 2017;5(2):2139-45. https://doi.org/10.21474/IJAR01/3387
30. Abdul Razak MK, Sultan AA. The importance of measurement of plasma fibrinogen level among patients with type- 2 diabetes mellitus. Diabetes Metab Syndr. 2019;13(2):1151-8. https://doi.org/10.1016/j.dsx.2019.01.049 PMid:31336458
31. Fluger I, Maderova K, Simek M, Hajek R, Zapletalova J, Lonsky V. Comparison of functional fibrinogen assessment using thromboelastography with the standard von Clauss method. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2012;156:260-1. https://doi.org/10.5507/bp.2011.035 PMid:22660203
32. Kautzky-Willer A, Leutner M, Harreiter J. Sex differences in type 2 diabetes. Diabetologia. 2023;66(6):986-1002. https://doi.org/10.1007/s00125-023-05891-x PMid:36897358 PMCid:PMC10163139
33. Getu F, Alemayehu E, Tesfaye A, Genanew B, Walle M. The evaluation of prothrombin time and activated partial thromboplastin time among diabetic and healthy controls in Africa: Systematic review and meta-analysis. Front Med. 2024;11:1445031. https://doi.org/10.3389/fmed.2024.1445031 PMid:39712180 PMCid:PMC11658991
34. Bryk-Wiązania AH, Undas A. Hypofibrinolysis in type 2 diabetes and its clinical implications: From mechanisms to pharmacological modulation. Cardiovasc Diabetol. 2021;20(1):191. https://doi.org/10.1186/s12933-021-01372-w PMid:34551784 PMCid:PMC8459566
35. Ząbczyk M, Ariëns RAS, Undas A. Fibrin clot properties in cardiovascular disease: From basic mechanisms to clinical practice. Cardiovasc Res. 2023;119(1):94-111. https://doi.org/10.1093/cvr/cvad017 PMid:36662542 PMCid:PMC10377755
36. Nencini F, Bettiol A, Argento FR, et al. Post-translational modifications of fibrinogen: Implications for clotting, fibrin structure and degradation. Mol Biomed. 2024;5:45. https://doi.org/10.1186/s43556-024-00214-x PMid:39477884 PMCid:PMC11525374
37. Mossberg K, Olausson J, Fryk E, Jern S, Jansson P-A, Brogren H. The role of the platelet pool of Plasminogen Activator Inhibitor-1 in well-controlled type 2 diabetes patients. PLoS One. 2022;17(8):e0267833. https://doi.org/10.1371/journal.pone.0267833 PMid:36044519 PMCid:PMC9432754