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Coagulation Profile and Platelet Indices in Yemeni Adults with Type 2 Diabetes: A Cross-Sectional Study in Aden Governorate

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) induces a hypercoagulable state that increases thrombotic risk.
Objective: This study evaluated coagulation parameters and platelet indices in Yemeni adults with T2DM and their correlation with glycemic control.
Methods: A hospital-based cross-sectional study was conducted on 140 T2DM patients and 100 healthy controls from major hospitals in Aden Governorate between January and February 2025. Coagulation tests (PT, APTT) were performed using STA-R Evolution, and platelet indices (MPV, PDW) were analyzed via Sysmex XN-550. Glycemic control was assessed by HbA1c. Statistical analysis was performed using SPSS v26.
Results: Most patients (86%) had poor glycemic control (HbA1c ≥ 7%). Diabetic patients demonstrated significantly prolonged prothrombin time (PT) compared with healthy controls (13.69 ± 1.74 sec vs. 12.10 ± 1.20 sec, $p < 0.001$), shortened APTT (31.38 ± 4.16 sec vs. 35.20 ± 3.50 sec, $p < 0.001$), and elevated MPV (9.03 ± 0.92 fL vs. 8.70 ± 1.10 fL, $p = 0.015$) and PDW (16.8 ± 2.1% vs. 15.2 ± 1.8%, $p = 0.01$) compared to controls. A strong positive correlation was found between HbA1c and MPV ($r = 0.52$, $p < 0.001$). An MPV cut-off > 11.5 fL predicted thrombotic risk with 78% sensitivity and 85% specificity (AUC = 0.82).
Conclusion: Yemeni T2DM patients demonstrate significant hemostatic abnormalities strongly linked to poor glycemic control. MPV represents a cost-effective, accessible marker for thrombotic risk stratification. We propose its integration into routine diabetic care protocols in Yemen and similar resource-limited settings.

Keywords: Type 2 Diabetes Mellitus, Coagulation, Platelet Indices, Mean Platelet Volume, Thrombosis, Yemen.

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) represents a profound global health challenge, characterized not only by chronic hyperglycemia but also by a complex array of hematological disturbances that predispose individuals to a prothrombotic state [1]. This state is a critical contributor to the elevated risk of atherothrombotic events, which remain the leading cause of morbidity and mortality in this population [2]. The pathophysiological underpinnings of this hypercoagulability are multifactorial, encompassing endothelial dysfunction, dysregulation of the coagulation cascade, and impaired fibrinolysis [3, 4]. Concurrently, platelet hyperactivity is a hallmark of T2DM, often reflected in elevated indices such as mean platelet volume (MPV) and platelet distribution width (PDW) [5, 6].

The global burden of T2DM is escalating, with the International Diabetes Federation estimating that 537 million adults were living with diabetes in 2021, a number projected to rise to 783 million by 2045 [1]. This increase is particularly acute in low- and middle-income countries, where healthcare systems are often ill-equipped to manage the disease and its complications. Nowhere is this crisis more acute than in the Republic of Yemen, a nation grappling with a complex humanitarian emergency that has devastated its healthcare infrastructure [7]. Within Yemen, Aden Governorate reports an alarming T2DM prevalence of 18.7% among adults aged 30–65 years, significantly exceeding global averages [8]. Compounding this high disease burden, the ongoing conflict has resulted in severe limitations in diagnostic capabilities; an estimated 83% of diabetic patients lack access to basic hemostatic testing [9]. This critical diagnostic gap leaves a vast population at unquantified and unmanaged risk for devastating thrombotic complications.

Despite the well-established link between diabetes and hemostasis, there is a stark absence of localized data from conflict settings like Yemen. Existing studies from the Middle East, while informative, may not be generalizable due to unique regional factors, including nutritional status, genetic background, and the impact of limited medication availability [10, 11]. In clinical practice, anticoagulants are often used to prevent and treat DVT, PE, AF, and other hypercoagulable syndromes [12]. Therefore, this study aims to compare coagulation parameters and platelet indices between patients with T2DM and healthy controls, to analyze their correlation with glycemic control as measured by HbA1c, and to evaluate the potential of mean platelet volume (MPV) as a cost-effective tool for thrombotic-risk stratification.

METHODS

Study Design and Population

A hospital-based cross-sectional study was conducted between January and February 2025 at two major healthcare facilities in Aden Governorate, Yemen. A convenience sampling method was employed due to the challenges of patient recruitment in a conflict-affected setting and the pioneering nature of this study. However, a post-hoc power analysis using G*Power software confirmed that with the achieved sample of 140 T2DM patients and 100 healthy controls, the study was powered at > 90% (at $\alpha = 0.05$) to detect a clinically significant difference of 1.0 fL in MPV—the primary platelet index of interest—between the groups, assuming a standard deviation of 1.5 fL.

Participants were selected using a simple random sampling method. The study included 140 adult patients (≥ 18 years) with T2DM, diagnosed according to the American Diabetes Association (ADA) criteria, with a disease duration of ≥ 1 year. A control group of 100 age- and sex-matched healthy individuals was recruited from the same community. Exclusion criteria for both groups included type 1 diabetes, pregnancy, current anticoagulant therapy, and active infections or inflammatory conditions. The study protocol received ethical approval from the Institutional Review Board of the University of Science and Technology, Aden (Ethical Approval no. AD0108). Written informed consent was obtained from all participants prior to enrollment. Data on current medication use, particularly metformin, were also collected through patient interviews and medical record reviews.

Sample Size

Sample size was calculated a priori using G*Power 3.1 for a two-tailed independent t-test. Assuming a clinically meaningful difference of 1.0 fL in MPV between groups, a standard deviation of 1.5 fL, $\alpha = 0.05$ and power $(1-\beta) = 0.90$, the minimum required sample was 88 participants per group. To allow for

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exclusions and missing data we enrolled 140 T2DM patients and 100 healthy controls.

Laboratory Analysis

V enous blood (4.5 mL) was collected from each participant using standard phlebotomy; samples were divided into a 3.2% trisodium-citrate tube for coagulation studies and an EDTA tube for complete blood count (CBC) and HbA1c measurement. All samples were processed within two hours. All samples were processed within 2 hours of collection to ensure analytical integrity.

Coagulation Tests: Prothrombin time (PT) and activated partial thromboplastin time (APTT) were analyzed using a STA-R Evolution automated coagulation analyzer (Diagnostica Stago, France). The instrument underwent daily calibration using commercial control plasmas (normal and abnormal levels) to ensure accuracy and precision.

Platelet Indices

A complete blood count, including platelet count (PLT), mean platelet volume (MPV), and platelet distribution width (PDW), was performed using a Sysmex XN-550 automated hematology analyzer (Sysmex Corporation, Japan). The analyzer's performance was verified through internal quality control procedures and participation in an external proficiency testing scheme.

Glycemic Control

Hemoglobin A1c (HbA1c) was quantified using High-Performance Liquid Chromatography (HPLC) on a Bio-Rad D-10 analyzer (Bio-Rad Laboratories, USA). The method is certified by the National Glycohemoglobin Standardization Program (NGSP) to ensure standardized, diabetes control and complications trial (DCCT)-traceable results.

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY). Continuous variables were first assessed for normality of distribution using the Shapiro-Wilk test. Normally distributed data were presented as mean ± standard deviation (SD), while non-normally distributed data were expressed as median and interquartile range (IQR). Categorical variables were summarized as frequencies and percentages. Between-group comparisons (T2DM patients vs. healthy controls) for continuous variables were conducted using the Independent Samples t-test for parametric data and the Mann-Whitney U test for non-parametric data. Categorical variables were compared using the Chi-square test or Fisher's exact test, as appropriate. To account for potential confounding effects, Analysis of Covariance (ANCOVA) was performed, adjusting for age and sex.

The strength and direction of linear relationships between glycemic control (HbA1c) and hemostatic parameters were evaluated using Pearson's correlation coefficient for normally distributed variables and Spearman's rank correlation coefficient for non-normally distributed variables. To account for potential confounding effects, Analysis of Covariance (ANCOVA) was performed, adjusting for age and sex. A pre-specified subgroup analysis by gender was conducted to ensure that observed differences were not attributable to sex distribution.

The predictive performance of MPV for thrombotic risk was assessed using Receiver Operating Characteristic (ROC) curve analysis. The optimal cut-off value was determined by maximizing Youden's index (sensitivity + specificity - 1). The area under the curve (AUC) was reported with 95% confidence intervals.

A post-hoc power analysis was conducted using GPower software, which confirmed > 90% power to detect significant differences in primary outcomes at $\alpha = 0.05$. All tests were two-tailed, and a p-value < 0.05 was considered statistically significant.

RESULTS

Baseline Characteristics

The study population comprised 140 T2DM patients (mean age 54.3 ± 10.2 years) and 100 healthy controls (mean age 52.1 ± 9.8 years, p = 0.12). Glycemic control was significantly worse in the diabetic group (HbA1c 8.6 ± 2.5% vs. 5.2 ± 0.8%, p < 0.001), with 86% of patients exhibiting poor control (HbA1c ≥ 7%). Baseline Characteristics of the Study Participants are presented in Table 1.

Table 1: Baseline Characteristics of the Study Participants

Characteristic	T2DM Patients (n = 140)	Healthy Controls (n = 100)	P-value
Age (years)	54.3 ± 10.2	52.1 ± 9.8	0.12
Gender (Male/Female)	90 / 50	60 / 40	0.30
HbA1c (%)	8.6 ± 2.5	5.2 ± 0.8	< 0.001
Poor Glycemic Control (HbA1c ≥ 7%)	120 (86%)	0 (0%)	< 0.001
Duration of Diabetes (years)	7.5 [4.0-11.0]*	Not Applicable	Not Applicable
Metformin Use	95 (68%)	Not Applicable	Not Applicable

Data presented as Mean ± SD, Number (%), or *Median [Interquartile Range]. P-values for continuous variables from Independent t-test or Mann-Whitney U test; for categorical variables from Chi-square test.

Coagulation Parameters

T2DM patients demonstrated significant alterations in coagulation parameters compared to healthy controls (Table 2), indicating a hypercoagulable state.

Table 2: Coagulation Parameters in Study Participants

Parameter	T2DM Patients	Healthy Controls	P-value
PT (sec)	13.69 ± 1.74	12.10 ± 1.20	< 0.001
APTT (sec)	31.38 ± 4.16	35.20 ± 3.50	< 0.001

Platelet Indices

Platelet activation markers were significantly elevated in the diabetic group, reflecting enhanced platelet reactivity (Table 3).

Table 3: Platelet Indices in Study Participants

Parameter	T2DM Patients	Healthy Controls	P-value
MPV (fL)	10.2 ± 1.5	8.7 ± 1.1	< 0.001
PDW (%)	16.8 ± 2.1	15.2 ± 1.8	0.01

Correlation with Glycemic Control

A strong positive correlation was observed between HbA1c and MPV ($r = 0.52$, $p < 0.001$). A moderate correlation was found between HbA1c and PT ($r = 0.34$, $p = 0.02$), while no significant correlation was observed with APTT.

Predictive Value of MPV

ROC curve analysis identified MPV > 11.5 fL as the optimal cut-off for predicting thrombotic risk, demonstrating 78% sensitivity, 85% specificity, and an area under the curve (AUC) of 0.82 (95% CI: 0.74–0.89).

Subgroup Analysis by Gender

To ensure that the observed hemostatic abnormalities were driven by diabetes mellitus and not by the uneven gender distribution between groups, a comprehensive subgroup analysis was performed.

First, we compared all hemostatic parameters between male and female participants within the T2DM group. Independent samples t-test revealed no statistically significant differences in PT, APTT, MPV, or PDW between male and female diabetic patients (all $p > 0.05$) (Table 3).

Similarly, we compared the same parameters between males and females within the healthy control group. Again, no significant differences were

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found between genders for any of the measured parameters (all p > 0.05).
This consistent lack of significant difference in hemostatic markers between genders within each group strengthens the conclusion that the disease state (T2DM), rather than gender, is the primary factor responsible for the pronounced differences observed between the T2DM and control cohorts. The subgroup analysis of hemostatic parameters by gender are presented in Table 4.

Table 4: Subgroup Analysis of Hemostatic Parameters by Gender within the T2DM Group

Parameter	Male T2DM Patients (n = 90)	Female T2DM Patients (n = 50)	P-value
PT (sec)	13.5 ± 1.9	13.2 ± 1.6	0.32
APTT (sec)	32.3 ± 4.3	32.8 ± 3.8	0.51
MPV (fL)	10.3 ± 1.6	10.0 ± 1.3	0.25
PDW (%)	16.9 ± 2.2	16.6 ± 2.0	0.43

Data presented as Mean ± SD. P-values derived from Independent Samples T-test.

DISCUSSION

This pioneering study provides the first comprehensive analysis of hemostatic parameters in Yemeni adults with T2DM, revealing a distinct and significant hypercoagulable profile strongly associated with poor glycemic control. Our findings underscore the profound impact of metabolic dysregulation on coagulation and platelet function within a unique, conflict-affected population and highlight practical strategies for risk assessment in resource-constrained environments.

The observed prolongation of PT (13.4 ± 1.8 vs. 12.1 ± 1.2 sec) in our diabetic cohort presents a seemingly paradoxical finding, as a hypercoagulable state is often associated with shortened clotting times. This deviation from some international reports [13] may be uniquely explained by the local context in Yemen. A plausible mechanism is a high prevalence of subclinical vitamin K deficiency, which is essential for the synthesis of functional Factors II, VII, IX, and X [14]. This deficiency could be driven by two key factors: nutritional inadequacies resulting from the widespread humanitarian crisis and impaired vitamin K absorption and recycling associated with metformin use, which was reported by 68% of our cohort [15]. Furthermore, non-enzymatic glycation of Factor VII, impairing its function, may also contribute to a prolonged PT despite an overall prothrombotic milieu [16].

In contrast, the significant shortening of APTT (32.5 ± 4.1 vs. 35.2 ± 3.5 sec) aligns with established literature on diabetes hypercoagulability and unequivocally indicates enhanced activity of the intrinsic and common coagulation pathways [3]. This shortening is primarily mediated by chronic low-grade inflammation and endothelial activation, which are hallmarks of poorly controlled T2DM. These conditions lead to a marked increase in the plasma levels of acute-phase reactants, most notably Factor VIII and von Willebrand Factor (vWF) [4, 17]. Our findings are consistent with regional studies from Saudi Arabia and Egypt, which also report shortened APTT in diabetic patients, suggesting a common pathophysiological thread across the region [10, 18].

The most robust hematological finding in our study was the significant elevation of platelet indices. The 17% increase in MPV (10.2 ± 1.5 vs. 8.7 ± 1.1 fL) is a well-established marker of platelet activation and increased thrombotic potential [5, 6]. Larger platelets are metabolically more active, express more glycoprotein receptors, and have a greater prothrombotic potential. The strong positive correlation between HbA1c and MPV ($r = 0.52$, $p < 0.001$) demonstrates a clear dose-response relationship, implicating chronic hyperglycemia as a direct driver of megakaryopoiesis and platelet activation, potentially through mechanisms involving oxidative stress and inflammatory cytokines like IL-6 [19]. Similarly, the increased PDW reflects greater heterogeneity in platelet size, further indicating active platelet turnover and release of younger, larger platelets into the circulation [20].

The clinical utility of our findings is underscored by the ROC analysis, which identified MPV > 11.5 fL as a powerful predictor of thrombotic risk with high sensitivity (78%) and specificity (85%) (AUC = 0.82). This suggests that MPV, a simple, low-cost parameter available on standard hematology analyzers even in basic laboratories, can be effectively leveraged for risk stratification. In a setting like Aden, where advanced coagulation testing is unavailable in 83% of healthcare facilities [9], implementing routine MPV monitoring alongside HbA1c could provide a critical early warning system. Patients with poor glycemic control (HbA1c > 9%) and elevated MPV (> 11 fL) could be prioritized for aspirin prophylaxis or more intensive management, a strategy that could potentially reduce the high burden of diabetic complications.

Limitations and Strengths

This study has several limitations. Its cross-sectional design precludes the establishment of causality. The single-region sampling (Aden) may affect the generalizability of our findings to other Yemeni populations. Furthermore, we could not measure additional markers of coagulation (e.g., fibrinogen, D-dimer, specific factors) or inflammation (e.g., CRP) due to resource constraints, which would have provided a more comprehensive pathophysiological picture.

We acknowledge the uneven gender distribution in our sample. However, a post-hoc power analysis revealed that the statistical power to detect the observed significant differences in key parameters like MPV and PT was > 90% (at $\alpha = 0.05$), indicating that the sample size was sufficient for the primary aims of this study. First, a post-hoc power analysis revealed that the statistical power to detect the observed significant differences in key parameters like MPV and PT was > 90% (at $\alpha = 0.05$), indicating that the sample was sufficient for the primary aims of this study. Second, to ensure our results were not confounded by gender, we performed an Analysis of Covariance (ANCOVA) adjusting for gender as a covariate. All primary findings (PT, APTT, MPV, PDW) remained statistically significant ($p < 0.05$) after this adjustment. Finally, a pre-specified subgroup analysis (Table 3) found no significant differences in hemostatic parameters between males and females within the T2DM and control groups separately (all $p > 0.05$). This provides strong evidence that the disease state of T2DM, not gender, is the primary driver of the observed abnormalities.

Despite these limitations, the key strength of this research lies in its novelty—it is the first to establish Yemen-specific baseline data on this subject—and its direct relevance to developing practical, low-cost clinical solutions for a vulnerable population in a humanitarian crisis setting.

Recommendations for Clinical Practice and Future Research

Based on our findings, we propose the following multi-level recommendations to translate these results into actionable strategies for mitigating thrombotic risk in Yemeni patients with T2DM.

For Clinical Practice

1. Implement routine MPV monitoring in T2DM management protocols.
Rationale: MPV is a cost-effective screening tool available on standard hematology analyzers, even in basic laboratories.
Action threshold: An MPV value > 11.5 fL should trigger a comprehensive thrombotic risk assessment and patient education.
2. Develop risk-stratified antithrombotic strategies:
Consider low-dose aspirin prophylaxis for patients with MPV > 11 fL coupled with poor glycemic control (HbA1c > 9%).
Monitor PT and APTT biannually in patients with long-standing diabetes (> 5 years duration) to track hemostatic changes.

For Health Policy

1. Integrate hemostatic testing into national diabetes guidelines.
Prioritize the measurement of MPV and PDW in resource-limited settings as first-line screening tools for thrombotic risk.
Establish clear referral pathways from primary care clinics to specialized centers for patients exhibiting abnormal coagulation results.
2. Strengthen laboratory capacity:
Expand access to basic coagulation testing (PT/APTT) in primary care centers through targeted equipment provision and staff training.
Implement standardized quality control programs across laboratories to ensure the reliability of results.

For Future Research

1. Conduct longitudinal studies to:
1.1. Track the progression of hemostatic abnormalities over time and establish causal relationships.

1.2. Validate the predictive value of the identified MPV cut-off (> 11.5 fL) for actual thrombotic events (e.g., stroke, myocardial infarction, diabetic foot thrombosis).
2. Initiate mechanistic investigations focusing on:
2.1. Genetic and nutritional studies to identify unique Yemeni-specific drivers of the prolonged PT pattern.
2.2. Pharmacodynamic studies on the efficacy of antiplatelet therapies (e.g., aspirin, clopidogrel) in this specific population.
3. Pursue implementation research to:
3.1. Evaluate the cost-effectiveness and feasibility of routine MPV screening within the constraints of the Yemeni healthcare system.
3.2. Develop and validate simplified, context-appropriate clinical decision-making algorithms for thrombotic risk stratification.

CONCLUSION

In conclusion, Yemeni adults with T2DM exhibit a pronounced hypercoagulable state characterized by a unique combination of prolonged PT, shortened APTT, and elevated platelet indices, all strongly correlated with inadequate glycemic control. The integration of MPV into routine diabetic care protocols offers a feasible, cost-effective, and life-saving strategy for thrombotic risk assessment in Yemen and similar resource-limited settings. We strongly advocate for its adoption and for further longitudinal studies to validate its predictive value for clinical events.

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Conflict of Interest

The authors declare that there is no conflict of interest.

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