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Research Article

Assessment of the Impact of Obesity on Coagulation Profile and D-Dimer Levels among Obese Adults in Shendi Locality, Sudan: A Case-Control Study

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Abstract

Background: Obesity is a major global health concern and an established independent risk factor for venous thromboembolism (VTE). Excess adipose tissue promotes chronic low-grade inflammation, endothelial dysfunction, increased coagulation activity, and impaired fibrinolysis, resulting in a hypercoagulable state. This study aimed to evaluate the effect of obesity on routine coagulation parameters and D-dimer levels among obese individuals in Shendi Locality, Sudan.

Methods: A case-control study was conducted in Shendi Locality, River Nile State, Sudan, between September and December 2025. The study included 100 participants, comprising 70 obese individuals (case group) and 30 apparently healthy individuals (control group). Venous blood samples were collected to determine prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (APTT), and D-dimer levels. Demographic characteristics, including age, sex, body mass index (BMI) classification, and family history of obesity, were recorded. Statistical analyses were performed using the independent samples t-test and one-way ANOVA, with statistical significance set at $p < 0.05$.

Results: Obese participants demonstrated significantly shorter PT (11.08 ± 1.87 vs. 13.54 ± 1.37 seconds, $p < 0.001$), lower INR (0.78 ± 0.14 vs. 0.96 ± 0.10, $p < 0.001$), and shorter APTT (15.85 ± 7.16 vs. 28.78 ± 5.53 seconds, $p < 0.001$) compared with the control group. In addition, D-dimer levels were significantly higher in obese individuals than in healthy controls (1.45 ± 2.53 vs. 0.17 ± 0.80 µg/L, $p = 0.007$). No statistically significant differences in coagulation parameters were observed according to sex, age, or family history of obesity. However, PT and INR differed significantly across BMI classes ($p = 0.003$ for both), whereas APTT and D-dimer showed no significant variation.

Conclusion: Obesity is associated with a hypercoagulable state characterized by shortened coagulation times and elevated D-dimer levels. These findings suggest that increasing adiposity contributes to coagulation activation and may increase thrombotic risk. Routine assessment of coagulation parameters may therefore be valuable in the clinical evaluation and management of obese individuals.

Keywords: Obesity; Coagulation profile; PT; APTT; INR; D-dimer; Hypercoagulability; BMI.

Introduction

Obesity is a chronic, multifactorial, and relapsing disease characterized by excessive accumulation of adipose tissue that adversely affects health. It develops through a complex interaction of genetic, metabolic, behavioral, environmental, and socioeconomic factors that disrupt the balance between energy intake and expenditure. The prevalence of obesity has increased dramatically worldwide over recent decades, making it one of the most important public health challenges of the twenty-first century. Obesity substantially increases the risk of numerous chronic diseases, including type 2 diabetes mellitus, cardiovascular disease, hypertension, dyslipidemia, certain malignancies, and metabolic syndrome. Consequently, effective management requires long-term lifestyle modification, pharmacological therapy, and, when appropriate, bariatric surgery to reduce morbidity and mortality [1]. Beyond its metabolic consequences, obesity is increasingly recognized as a disorder of chronic low-grade inflammation. Adipose tissue functions as an active endocrine organ that secretes numerous bioactive molecules, including adipokines, cytokines, and inflammatory mediators. Excess adiposity promotes persistent inflammatory activation, endothelial dysfunction, oxidative stress, and metabolic disturbances that contribute to vascular injury and increased cardiovascular risk. These pathophysiological alterations also influence the hemostatic system by promoting coagulation activation and suppressing fibrinolysis, thereby increasing the risk of thrombotic events [2].

Hemostasis is a tightly regulated physiological process that maintains the balance between bleeding and thrombosis. Following vascular injury, exposure of tissue factor (TF) initiates the coagulation cascade through activation of the extrinsic pathway, ultimately leading to thrombin generation and fibrin clot formation. Contemporary understanding recognizes coagulation as a complex network of interacting cellular and plasma components rather than the traditional intrinsic and extrinsic pathway model alone. Laboratory evaluation of coagulation commonly includes prothrombin time (PT), activated partial thromboplastin time (APTT), international normalized ratio (INR), thrombin time (TT), fibrin degradation products (FDPs), and D-dimer measurements, all of which provide valuable information regarding different aspects of the coagulation process [3]. D-dimer is a specific degradation product generated during plasmin-mediated breakdown of cross-linked fibrin. Elevated plasma D-dimer concentrations indicate activation of both coagulation and fibrinolysis and are widely used in the diagnosis and exclusion of venous thromboembolism (VTE), including deep vein thrombosis (DVT) and pulmonary embolism (PE). Increased D-dimer levels are also observed in disseminated intravascular coagulation (DIC), malignancy, severe infections, inflammatory disorders, trauma, postoperative conditions, pregnancy, and cardiovascular diseases. Several laboratory methods are available for D-dimer measurement, including enzyme-linked immunosorbent assay (ELISA), latex-enhanced immunoturbidimetric assays, and fluorescence-based point-of-care tests, with ELISA remaining the reference method because of its high diagnostic sensitivity [4-6]. Accumulating evidence indicates that obesity is associated with a hypercoagulable state resulting from increased synthesis of procoagulant factors, including fibrinogen, factor VII, factor VIII, von Willebrand factor, and plasminogen activator inhibitor-1 (PAI-1), together with impaired fibrinolytic activity. These alterations promote excessive thrombin generation and fibrin formation while reducing fibrin degradation, thereby increasing susceptibility to venous thromboembolism and other cardiovascular complications. Consequently, obese individuals frequently exhibit higher baseline D-dimer concentrations than normal-weight individuals, which may reduce the specificity of D-dimer testing for acute thrombotic events in this population [7]. Although several international studies have demonstrated an association between obesity and coagulation abnormalities, data from Sudan remain limited. Moreover, the relationship between obesity severity, coagulation parameters, and D-dimer levels has not been adequately investigated among Sudanese adults. Therefore, this study aimed to evaluate the effect of obesity on PT, INR, APTT, and D-dimer levels among obese adults in Shendi Locality, River Nile State, Sudan, and to examine the association of these parameters with body mass index, age, sex, and family history of obesity.

2. Materials and Methods

Study Design

A case-control study was conducted to evaluate the impact of obesity on coagulation profile parameters and D-dimer levels among obese adults in Shendi Locality, River Nile State, Sudan.

Study Area and Period

The study was carried out in Shendi Locality, River Nile State, Sudan, over four months from September to December 2025.

Study Population

The study included 100 participants, comprising 70 obese individuals who served as the case group and 30 apparently healthy individuals who served as the control group. Obese participants were recruited based on their body mass index (BMI), while healthy volunteers with normal BMI and no known coagulation disorders were enrolled as controls.

Inclusion Criteria

The study included adults diagnosed with obesity (BMI ≥30 kg/m²) who agreed to participate and provided informed consent. Healthy individuals with normal body weight and without known systemic diseases were included in the control group.

Exclusion Criteria

Participants were excluded if they had any of the following conditions:

- Pregnancy.
- Current anticoagulant therapy.
- History of venous thromboembolism.
- Chronic liver disease.
- Chronic kidney disease.
- Active malignancy.
- Acute inflammatory or infectious diseases.
- Known inherited or acquired bleeding disorders.

Data Collection

Demographic and clinical information was collected using a structured questionnaire.

The recorded variables included:

- Age
- Sex
- Body mass index (BMI)
- BMI classification
- Family history of obesity

BMI was calculated using the standard formula:
BMI (kg/m²) = Weight (kg) / Height² (m²)
Obese participants were classified according to the World

Health Organization (WHO) BMI classification:

- Class I obesity: 30.0–34.9 kg/m²
- Class II obesity: 35.0–39.9 kg/m²
- Class III obesity: ≥40.0 kg/m²

Sample Collection

Approximately 3 mL of venous blood was collected from each participant by standard venipuncture into sodium citrate anticoagulant tubes (3.2% sodium citrate). The samples were gently mixed immediately after collection to ensure proper anticoagulation.
Plasma was separated by centrifugation according to the manufacturer's recommendations and analyzed without unnecessary delay to preserve coagulation factor activity.

Laboratory Analysis

The following coagulation parameters were measured:

- Prothrombin Time (PT)
- International Normalized Ratio (INR)
- Activated Partial Thromboplastin Time (APTT)
- D-dimer concentration

All laboratory analyses were performed according to the manufacturer's instructions using standard operating procedures under appropriate quality control measures.

Statistical Analysis

Data were entered, cleaned, and analyzed using the Statistical Package for the Social Sciences (SPSS) software (IBM SPSS Statistics, Version XX). Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Comparisons between obese participants and healthy controls were performed using the independent samples t-test. Comparisons among BMI classes were conducted using one-way analysis of variance (ANOVA). A p-value < 0.05 was considered statistically significant.

3. Result

A total of 100 participants were enrolled in this case-control study, including 70 obese individuals (70%) in the case group and 30 apparently healthy individuals (30%) in the control group (Table 1). Among the obese participants, females represented the majority (48, 67%), whereas males accounted for 22 (33%). In the control group, 17 (57%) were females and 13 (43%) were males (Table 2). According to the WHO BMI classification, obese participants were categorized into Class I (n = 19), Class II (n = 15), and Class III (n = 36) obesity, with mean BMI values of 32.94 ±, 40.37 ±, and 51.62 ± kg/m², respectively (Table 3). Regarding age distribution, 36 (52%) obese participants were younger than 40 years, whereas 34 (48%) were aged 40 years or older. A similar age distribution was observed in the control group (Table 4). The obese group demonstrated significantly altered coagulation parameters compared with the healthy control group. The mean PT was significantly shorter in obese participants than in controls (11.08 ± 1.87 vs. 13.54 ± 1.37 seconds, p < 0.001). Likewise, the mean INR was significantly lower in the obese group (0.78 ± 0.14 vs. 0.96 ± 0.10, p < 0.001). Similarly, obese participants exhibited a significantly shorter APTT than healthy controls (15.85 ± 7.16 vs. 28.78 ± 5.53 seconds, p < 0.001). Furthermore, serum D-dimer concentrations were significantly higher among obese individuals than among healthy controls (1.45 ± 2.53 vs. 0.17 ± 0.80 µg/L, p = 0.007) (Table 5). No statistically significant differences were observed in PT, INR, APTT, or D-dimer levels

between male and female obese participants. The mean PT was 11.27 seconds in males and 10.97 seconds in females (p = 0.522). Likewise, INR, APTT, and D-dimer values did not differ significantly between the two sexes (p > 0.05) (Table 6). When obese participants were stratified by age, no statistically significant differences were found in PT, INR, APTT, or D-dimer levels. Participants younger than 40 years and those aged 40 years or older showed comparable coagulation profiles, with all comparisons yielding p-values greater than 0.05 (Table 7). Analysis according to obesity class revealed statistically significant differences in PT and INR among BMI categories. PT progressively decreased from 11.97 seconds in Class I obesity to 10.38 seconds in Class III obesity, with a statistically significant difference (p = 0.003). Similarly, INR showed a significant decline across increasing BMI classes (p = 0.003). In contrast, neither APTT (p = 0.260) nor D-dimer (p = 0.077) differed significantly among BMI categories (Table 8). No statistically significant association was observed between family history of obesity and coagulation parameters. Participants with and without a family history of obesity exhibited comparable PT, INR, APTT, and D-dimer values, with all comparisons demonstrating p-values greater than 0.05 (Table 9).

Characteristic	Frequency	Percent %	
Study group	Case	70	70%
	Control	30	30%

Table 1. Distribution of case and control group.

Percent%	Frequency	Sex	Study group
33%	22	Male	Case
67%	48	Female	
43%	13	Male	Control
57%	17	Female	

Table 2. shows the distribution of obese and healthy individuals according to gender.

Study group	BMI class	No	Mean	P. value
Case	Class1	19	32.942	0.000
	Class2	15	40.373	
	Class3	36	51.622	

Table 3. Show the distribution case group according to BMI class.

Study group	Age	Frequency	Percent %
Case	Less than 40	36	52%
	More than 40	34	48%
Control	Less than 40	17	57%
	More than 40	13	43%

Table 4. Show the distribution of individuals in the case and control groups according to age.

Group		No	Mean	SD	P. value
PT/sec	Case	70	11.079	1.8678	0.000
	Control	30	13.543	1.3655	
APTT/ sec	Case	70	15.847	7.1585	0.000
	Control	30	28.777	5.5323	
INR	Case	70	0.7789	0.13641	0.000
	Control	30	0.9602	0.9988	

Table 5. Show the mean of PT/sec, INR, APTT/sec and D-dimer in case and control groups.

Group	Gender	No	Mean	P. value
PT/sec	Male	25	11.272	0.522
	Female	45	10.971	
APTT/sec	Male	25	16.676	0.474
	Female	45	15.387	
INR	Male	25	0.7924	0.541
	Female	45	0.7714	
D-dimer	Male	25	1.428	0.144
	Female	45	1.462	

Table 6. Show the mean of PT, INR, APTT and D-dimer in test group according to gender.

Group	Age	No	Mean	P. value
PT/sec	Less than 40	36	10.872	0.352
	More than 40	34	11.294	
APTT/sec	Less than 40	36	15.878	0.971
	More than 40	34	15.815	

INR	Less than 40	36	0.7642	0.357
	More than 40	34	0.7945	
D-dimer	Less than 40	36	1.0192	0.144
	More than 40	34	1.9056	

Table 7. Show the mean of PT, INR, APTT, and D-dimer in the test group according to age.

Group	Class	Mean	P. value
PT/sec	Class1	11.968	0.003
	Class2	11.640	
	Class3	10.375	
	Class1	18.137	
APTT/sec	Class2	15.333	0.260
	Class3	14.853	
INR	Class1	0.8446	0.003
	Class2	0.8215	
	Class3	0.7265	
D-dimer	Class1	2.0932	0.077
	Class2	2.2213	
	Class3	0.7886	

Table 8. Show the mean of PT, INR, APTT, and D-dimer in the test group according to body mass index.

Group	Family History	No	Mean	P. value
PT/sec	Yes	39	10.098	0.395
	No	31	11.294	
APTT/sec	Yes	39	15.562	0.711
	No	31	16.206	
INR	Yes	39	0.7671	0.420
	No	31	0.7938	
D-dimer	Yes	39	1.5969	0.589
	No	31	1.2264	

Table 9. Show the mean of PT, INR, APTT, and D-dimer in the test group according to family History of obesity.

4. Discussion

Obesity is increasingly recognized as a chronic inflammatory condition associated with profound alterations in hemostasis that predispose affected individuals to thrombotic complications. Excess adipose tissue promotes endothelial dysfunction, increased synthesis of procoagulant factors, impaired fibrinolysis, and persistent low-grade inflammation, all of which contribute to the development of a hypercoagulable state [8]. The present study evaluated the effects of obesity on routine coagulation parameters and D-dimer levels among obese adults in Shendi Locality, Sudan.

The findings demonstrated that obese participants had significantly shorter PT and lower INR values than healthy controls, indicating enhanced activation of the extrinsic coagulation pathway. Increased circulating concentrations of coagulation factors, particularly factor VII and fibrinogen, together with obesity-induced inflammatory cytokines, may accelerate thrombin generation and shorten coagulation times. These findings are consistent with those reported by Basaran et al. [9], who observed increased coagulation activity in obese individuals. A significantly shorter APTT was also observed in the obese group. This finding suggests increased activity of the intrinsic coagulation pathway and supports the concept that obesity is associated with a prothrombotic state. Elevated plasma concentrations of coagulation factors VIII, IX, XI, XII, and fibrinogen have previously been reported in obese individuals and may explain the shortened APTT observed in this study. Similar findings have been described by Ay et al. [10] and Bokari et al. [11], both of whom reported increased coagulation potential in obese populations. D-dimer concentrations were significantly higher in obese participants than in healthy controls. This observation reflects increased fibrin formation and degradation secondary to continuous activation of coagulation and fibrinolysis. Chronic inflammation associated with obesity stimulates tissue factor expression, enhances thrombin generation, and increases fibrin turnover, resulting in elevated baseline D-dimer concentrations even in the absence of clinically apparent thrombosis. Comparable findings have been reported by Campello et al. [12], Di Castelnuovo et al. [13], and Purdy et al. [14], all of whom demonstrated higher D-dimer levels among overweight and obese individuals.

The present study found no significant association between age and coagulation parameters among obese participants. This suggests that obesity itself may have a greater influence on coagulation status than age within the studied population. Routine coagulation assays primarily assess global coagulation function and may not detect subtle age-related hemostatic alterations in clinically stable obese individuals. Similar observations have been reported in previous studies [15]. Likewise, no statistically significant differences were identified between male and female participants regarding PT, INR, APTT, or D-dimer levels. Although sex hormones may influence individual coagulation factors, obesity-related inflammatory and metabolic changes appear to exert a stronger effect on overall coagulation status than biological sex. Consequently, the prothrombotic alterations associated with obesity may occur similarly in both men and women. When

participants were classified according to BMI, PT and INR differed significantly across obesity classes, whereas APTT and D-dimer did not. These findings suggest that increasing adiposity may predominantly affect the extrinsic coagulation pathway, while intrinsic coagulation activity and fibrinolytic markers remain relatively stable once obesity is established. The absence of significant differences in D-dimer across BMI classes may indicate that coagulation activation reaches a plateau in severe obesity or may reflect the limited sample size within each BMI subgroup. Furthermore, family history of obesity was not associated with significant differences in any of the investigated coagulation parameters. This finding suggests that acquired metabolic and inflammatory changes accompanying obesity have a greater influence on coagulation status than hereditary predisposition alone. Lifestyle factors, adipose tissue dysfunction, and obesity-related metabolic abnormalities are therefore likely to be the principal determinants of the observed hypercoagulable state. Overall, the present findings support accumulating evidence that obesity is associated with enhanced coagulation activity and increased fibrin turnover. These hemostatic alterations may contribute to the elevated risk of venous thromboembolism and other cardiovascular complications observed in obese individuals. Early laboratory assessment of coagulation parameters, together with effective weight reduction strategies, may therefore play an important role in identifying high-risk individuals and reducing future thrombotic events.

5. Limitations

Several limitations should be considered when interpreting the findings of this study. First, the relatively small sample size may have limited the statistical power to detect subtle associations. Second, the study was conducted at a single center, which may reduce the generalizability of the findings to other populations. Third, additional hemostatic biomarkers, including fibrinogen, factor VIII, von Willebrand factor, plasminogen activator inhibitor-1 (PAI-1), and inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6), were not evaluated. Finally, the case-control design identifies associations but cannot establish causal relationships between obesity and coagulation abnormalities.

6. Conclusion

The present study demonstrated that obesity is associated with significant alterations in coagulation parameters, characterized by shortened PT, INR, and APTT values, along with elevated D-dimer levels, compared with healthy individuals. These findings support the presence of a hypercoagulable state in obese adults and suggest that obesity contributes to increased thrombotic risk through activation of the coagulation system and enhanced fibrin turnover. Although coagulation parameters were not significantly influenced by age, sex, or family history of obesity, significant differences in PT and INR were observed across BMI categories, indicating that the severity of obesity may influence specific aspects of the coagulation process. Routine assessment of coagulation markers in obese individuals may therefore facilitate early identification of patients at increased risk of thrombotic complications and support timely preventive interventions.

7. Recomandations

Based on the findings of this study, further multicenter studies with larger sample sizes are recommended to confirm these observations in different populations. Future research should incorporate additional coagulation, fibrinolytic, inflammatory, and endothelial biomarkers to provide a more comprehensive understanding of obesity-associated hemostatic dysfunction. Longitudinal studies investigating the effects of weight reduction, lifestyle modification, and bariatric surgery on coagulation parameters are also warranted. Moreover, routine coagulation assessment may be considered in obese individuals, particularly those with severe obesity or additional cardiovascular risk factors, to facilitate early detection of thrombotic risk.

Consent

The patient's written consent has been collected

Ethical Aproval

Ethical approval was obtained from the appropriate institutional ethics committee before commencement of the study. Written informed consent was obtained from all participants before enrollment. All collected data were treated confidentially and used exclusively for research purposes in accordance with the principles of the Declaration of Helsinki.

Competing Intrest

Authors have declared that no competing interests exist.

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