بسم الله الرحمن الرحيم
Omdurman Islamic University Journal of Applied Sciences (OIUJ)-(ASC)
مجلة جامعة أم درمان الاسلامية للعلوم التطبيقية
https://journal.oiu.edu.sd/index.php/oiuj
https://doi.org/ 10.52981/oiujas.v21i3.3393
2025; 21(3)17-28
ISSN: 5361-1858

Vitamin D Deficiency, Dyslipidemia, and Deep Vein tThrombosis Susceptibility in Sudanese Women: A Case-Control Study

Marwan A. Abdullah[1], Fayza A. Rahamtalla[1], Abdelmula M. Abdalla[2]

[1]Department of clinical chemistry, Faculty of Medical Laboratory Sciences, Omdurman Islamic University, Omdurman, Sudan

[2] Department of Clinical Chemistry, Faculty of Medical Laboratory Sciences, Al-Neelain University, Khartoum, Sudan

To cite this article:

Marwan A. Abdullah, Fayza A. Rahamtalla, Abdelmula M. Abdalla, Vitamin D Deficiency, Dyslipidemia, and Deep Vein thrombosis Susceptibility in Sudanese Women: A Case-Control Study, Omdurman Islamic University Journal, Sudan

ISSN: 5361-1858

https://doi.org/ 10.52981/oiujas.v21i3.3393

Abstract

Deep vein thrombosis (DVT) is a clinically significant condition resulting from the formation of thrombi in the deep venous system, most commonly in the lower extremities. It poses substantial morbidity and mortality risks, particularly due to its potential progression to pulmonary embolism (PE), a life-threatening complication characterized by embolic migration of the thrombus to the pulmonary vasculature. The etiology of DVT is multifactorial, involving a complex interplay of genetic predispositions, acquired conditions, and environmental influences. Established risk factors include prolonged immobility, surgical interventions, malignancy, inherited thrombophilias, and hormonal fluctuations associated with pregnancy and the use of oral contraceptives [1].

In recent years, increasing attention has been directed toward the role of non-traditional risk factors, particularly those related to nutritional and metabolic status, in the pathogenesis of thromboembolic events. Among these, serum vitamin D concentration and lipid profile abnormalities have emerged as potential contributors. While vitamin D is conventionally recognized for its role in calcium homeostasis and bone metabolism, it also exhibits a range of pleiotropic effects, including modulation of immune responses, suppression of inflammatory pathways, and potential anticoagulant activity. Several studies have suggested an inverse relationship between vitamin D deficiency and thrombotic risk, possibly mediated through effects on endothelial function, inhibition of thrombogenic pathways, and enhancement of

fibrinolysis [2,3].Similarly, dyslipidemia manifested by elevated total cholesterol, low-density lipoprotein (LDL), triglycerides, and reduced high-density lipoprotein (HDL) cholesterol has been extensively studied in the context of atherosclerotic cardiovascular disease. However, its potential role in venous thromboembolism (VTE), including DVT, remains less clearly defined. Proposed mechanisms linking dyslipidemia to thrombogenesis include endothelial dysfunction, oxidative stress, platelet hyperactivity, and disruption of anticoagulant balance, all of which may predispose to venous clot formation [4,5]. Despite the global burden of DVT, there is a notable paucity of data examining these emerging risk factors within African populations. Sudanese women, in particular, may be subject to unique environmental exposures, nutritional deficiencies, and reproductive health challenges that modulate their thrombotic risk profile. However, evidence in this demographic remains scarce. Investigating the relationship between serum vitamin D status, lipid profiles, and the risk of DVT in this population is essential to inform context-specific screening, prevention, and management strategies. Accordingly, the present study aims to evaluate serum vitamin D and lipid parameters in Sudanese women and to elucidate their potential association with DVT risk.

Materials and Methods

Study Design and Population

This study was designed as a hospital-based case-control study. Participants were recruited from selected public maternity hospitals in Khartoum State, namely Omdurman Obstetric Hospital and Al-Saudi Hospital – Omdurman, as well as from obstetrics and gynecology clinics in Al-Burgaig, Northern State, during the period from May 2022 to May 2024.

Inclusion Criteria

The case group included pregnant and postpartum women attending the aforementioned hospitals for routine follow-up, who were diagnosed with deep vein thrombosis (DVT) during the study period using Doppler ultrasound. The control group consisted of pregnant and non-pregnant women without clinical or radiological signs of DVT, attending the same hospitals for routine follow-up. Controls were matched to cases based on age, gravidity, and sample size.

Sample size

The sample size has been calculated by computer program, according to provided, confidence level 1.96, standard deviation 1.5, and marginal error 0.4

$$N = \frac{Z\ value2 \times \partial 2}{e2}$$

N= Number of patients

Z value = 1.96 or 95%

a = SD = 1.5

e = Marginal errors = 0.4

$$N = \frac{(1.96)2 \times (1.5 \times 1.5)}{0.4 \times 0.4} = 55 \text{ for each group}$$

So the total sample size were 165 participants, 55 DVT patients, 55 healthy pregnant women and 55healthy non-pregnant women.

Data Collection and Blood Sampling

Data were collected through direct interviews with participants using a structured, coded questionnaire developed by the research team to obtain demographic and clinical information. Additional clinical and laboratory data were recorded on the same form. Approximately 5 mL of peripheral venous blood was drawn from each participant into plain tubes for the assessment of serum vitamin D and lipid profile. Samples were allowed to clot at room temperature for one hour, followed by centrifugation at 3000 rpm for 10 minutes. The resulting sera were stored at −20°C until laboratory analysis. Serum 25-hydroxyvitamin D3 levels were measured using an ELISA kit based on a competitive enzyme immunoassay technique (Fortress Diagnostics Limited, United Kingdom; Catalogue No: BXE0114A). Total cholesterol, triglycerides, and HDL-cholesterol were determined enzymatically using Bio Systems reagents and a spectrophotometer. LDL and VLDL levels were calculated using the Freidwald formula.

Data Management and Statistical Analysis

Descriptive statistics were used to summarize both qualitative and quantitative variables. Results were presented as mean ± standard deviation (SD). Comparisons between two groups were performed using the Student's t-test, while comparisons across more than two variables employed the chi-square test (χ^2). Pearson's correlation coefficient was calculated to evaluate associations between continuous variables, and binary logistic regression was applied to assess predictors and strength of association among studied factors. A P-value ≤ 0.05 was considered statistically significant. All statistical analyses were conducted using SPSS software (Version 24.0, IBM Corp., and Armonk, NY, USA) and results were presented in tabular format.

Results

1. General characteristics of the study subjects

Table 1 summarizes the demographic and clinical characteristics of the three study groups: healthy non-pregnant women (n = 55), healthy pregnant women (n = 55), and women diagnosed with Deep Vein Thrombosis (DVT) (n = 55). It presents the mean ± standard deviation for continuous variables (e.g., age, gravida), and frequencies with percentages for categorical variables such as contraceptive use, history of previous DVT, and anticoagulant therapy. Notably, mean age differed across groups: 34.6 ± 4.3 years in healthy non-pregnant women, 29.3 ± 3.2 years in healthy pregnant women, and 32.7 ± 6.8 years in DVT patients. Gravida, reported only in the pregnant group, had a mean of 2.95 ± 1.3. Contraceptive use was documented in both the pregnant (47.3% yes, 52.7% no) and DVT groups (50.9% yes, 49.1% no). A previous DVT event was reported in 63.6% of pregnant women and 69.1% of DVT patients. Anticoagulant usage was observed in 72.7% of women in both groups.

2. Comparison of vitamin D levels and lipids profile between the different study groups

Table 2 presents a comparative analysis of serum vitamin D levels and lipid profile parameters across the three groups. P-values were used to assess statistical significance: values <0.05 were considered significant (S), and <0.03 highly significant (HS). All comparisons for vitamin D and lipid parameters Total Cholesterol (T. Cho), Triglycerides (TG), HDL, LDL, and VLDL showed highly significant differences (P = 0.01 HS). Vitamin D levels were highest in healthy non-pregnant women (39.4 ± 4.7 ng/mL), followed by the DVT group (24.1 ± 3.7 ng/mL), and lowest in the pregnant group (19.5 ± 3.7 ng/mL). Conversely, pregnant women had the highest mean levels of T. Cho (175.2 ± 20.8 mg/dL), TG (157.1 ± 20.2 mg/dL), LDL (88.9 ± 18.4 mg/dL), and VLDL (31.4 ± 4.1 mg/dL). HDL levels were also higher in pregnant (54.9 ± 6.3 mg/dL) and healthy non-pregnant women (53.4 ± 8.4 mg/dL) compared to the DVT group (46.3 ± 10.5 mg/dL.

3. Comparative Analysis of Vitamin D and

Lipid Profiles in Sudanese Women: Focus on DVT and Pregnancy

Table: 3 presents a comparative overview of serum vitamin D levels and lipid profile components including Total Cholesterol (T. Cho), Triglycerides (TG), High-Density Lipoprotein (HDL), Low-Density Lipoprotein (LDL), and Very-Low-Density Lipoprotein (VLDL) among Sudanese women diagnosed with Deep Vein Thrombosis (DVT) and healthy pregnant women. The data are stratified based on history of previous DVT attacks, anticoagulant therapy, and contraceptive use. All values are presented as mean ± standard deviation, with significance assessed via P-values (NS: Not Significant, HS: Highly Significant).

1. Women with DVT

• Effect of Previous DVT Attack:

Women with a history of DVT attacks (n = 35) showed significantly lower vitamin D levels compared to those without prior attacks (n = 20): 23.0 ± 3.2 vs. 26.0 ± 3.8, P = 0.01 (HS). No significant differences were detected in lipid profile parameters (T. Cho, TG, HDL, LDL, VLDL) between the two groups.

• Effect of Anticoagulant Use:

No statistically significant differences were observed in vitamin D or lipid profile levels between women on anticoagulant therapy (n = 40) and those not receiving anticoagulants (n = 15).

2. Healthy Pregnant Women

• Effect of Previous DVT Attack:

Pregnant women with a history of DVT (n = 28) had slightly lower vitamin D levels than those without (n = 27), though the difference was not statistically significant: 19.2 ± 3.6 vs. 19.8 ± 3.9, P = 0.58 (NS). No significant alterations were observed in any lipid parameters between the groups.

• Effect of Anticoagulant Use:

Comparison between anticoagulant users (n = 38) and non-users (n = 17) revealed no statistically significant differences in either vitamin D or lipid levels.

• Effect of Contraceptive Use:

No significant differences were found in vitamin D or lipid profiles between pregnant women using contraceptives (n = 26) and those not using them (n = 29). The most notable finding from this analysis is the significantly lower vitamin D levels in women with DVT who had a previous thrombotic event, suggesting a potential association between vitamin D deficiency and recurrent thrombosis. However, no meaningful differences were detected in lipid profiles across any of the studied subgroups. These findings emphasize the possible role of vitamin D in thrombotic risk, particularly among women with a prior history of DVT

4. Correlation of study variables with gravida among healthy pregnant women

Table 4 presents the correlation coefficients (r) and associated p-values for the relationship between gravida and various biochemical parameters. A statistically significant inverse correlation was found between vitamin D levels and gravida (r = –0.266, P = 0.04 S), indicating that vitamin D levels tend to decline as parity increases. No significant correlations were observed between gravida and any lipid profile parameter (P>0.05 NS).

5. Logistic Regression Analysis of DVT Risk Factors

Table 6 reports the results of a logistic regression model evaluating potential biochemical predictors of DVT. The table includes the regression coefficient (B), standard error (SE), p-value, and odds ratio (Exp(B)). Among the variables assessed, only HDL showed a borderline significant association with DVT risk (B = –0.124, P = 0.05; OR = 0.883), suggesting a possible protective effect. All other variables including vitamin D, T. Cho, TG, LDL, and VLDL did not show statistically significant associations (P > 0.05).

Table 1: General characteristics of the

Parameters	Women with DVT (n=55)	Healthy pregnant (n=55)	Healthy non-pregnant(n =55)
Age (years) mean ± SD	32.7±6.8	29.3±3.2	34.6±4.3
Gravida Mean ± SD	-	2.95±1.3	-
Contraceptive usage: **Yes** **No**	- -	26 (47.3 %) 29 (52.7 %)	- -
DVTprevious attack: **Yes** **No**	35(63.6 %) 20(36.4 %)	28 (50.9 %) 27 (49.1 %)	- -
Anticoagulant usage: **Yes** **No**	40 (72.7 %) 15 (27.3 %)	38 (69.1 %) 17 (30.9 %)	- -

study subjects

- **DVT»** Deep vein thrombosis patients

Variables	Woman with DVT(n=55)	Healthy pregnant(n=55)	Healthy non-pregnant(n=55)	*P- value*
Vitamin D ng/ml	24.1±3.7	19.5±3.7	39.4±4.7	0.01^{HS}
T. Cholesterol mg/dl	161.0±29.4	175.2±20.8	148.0±15.9	0.01^{HS}
Tri glyceride mg/dl	152.9±24.3	157.1±20.2	132.2±16.3	0.01^{HS}
HDL mg/dl	46.3±10.5	54.9±6.3	53.4±8.4	0.01^{HS}
LDL mg/dl	81.7±23.5	88.9±18.4	67.9±11.0	0.01^{HS}
VLDL mg/dl	30.6±4.9	31.4±4.1	26.4±3.3	0.01^{HS}

Table 2: Comparison of vitamin D levels and lipids profile between the different study groups

- HS» highly significant (p value <0.01), HDL» high density lipoprotein,
- LDL» low density lipoprotein, VLDL» very low density lipoprotein

Table 3: Comparison of vitamin D and lipid profile levels based on the previous attack and anticoagulant usage in woman with DVT and healthy pregnant women

Variables	Vitamin D (ng/ml)	T. Cho (mg/dl)	TG (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	VLDL (mg/dl)
Women with DVT						
Previous attack:						
Previous attack (n =35)	23.0±3.2	160±31.4	152.4±24.8	47.7±9.8	80.1±25.4	30.5±4.9
No- attack (n = 20)	26.0±3.8	163±26.1	153.8±24.1	43.8±11.3	84.5±20.1	30.8±4.8
P- value	0.01^{HS}	0.70^{NS}	0.83^{NS}	0.18^{NS}	0.50^{NS}	0.83^{NS}
Anticoagulant usage:						
Usage (n =40)	24.0±3.2	160.5±31.0	153.8±24.3	47.4±9.7	81.5±24.4	30.8±4.8
Non- usage (n =15)	24.6±4.8	162.5±25.4	150.5±25.1	43.3±12.0	82.1±21.8	30.1±5.0
P- value	0.60^{NS}	0.81^{NS}	0.66^{NS}	0.19^{NS}	0.92 ^{NS}	0.66 ^{NS}
Healthy pregnant women						
Previous attack:						
Previous attack (n =28)	19.8±3.9	175.6±22.5	158.0±20.3	55.1±7.2	89.0±17.3	31.6±4.1
No- attack (n =27)	19.2±3.6	174.8±19.5	156.3±20.3	54.7±5.5	88.8±19.8	31.3±4.1
P- value	0.58^{NS}	0.89^{NS}	0.77^{NS}	0.85^{NS}	0.97^{NS}	0.77^{NS}
Anticoagulant usage:						
Usage (n =38)	18.9±3.9	175.3±20.6	157.2±21.1	54.6±6.6	89.4±17.9	31.5±4.2
Non- usage (n =17)	21.0±2.9	175.1±21.1	157.0±20.0	55.0±6.3	88.7±18.8	31.4±4.0
P- value	0.06 ^{NS}	0.97^{NS}	0.96^{NS}	0.82^{NS}	0.90^{NS}	0.96^{NS}
Contraceptive usage:						
Usage (n =26)	19.5±3.3	176.3±21.9	158.2±21.8	55.7±6.5	89.1±19.0	31.6±4.4
Non- usage (n =29)	19.6±4.1	174.2±20.1	156.1±18.9	54.2±6.2	88.7±18.1	31.2±3.9
P- value	0.91^{NS}	0.71^{NS}	0.71^{NS}	0.39^{NS}	0.94^{NS}	0.71^{NS}

Table 4: Correlation of study variables with gravida among healthy pregnant women

Variables	Coefficient	
	r	*P- value*
Vitamin D ng/ml	-0.266	0.04^{S}
T. Cholesterol mg/dl	-0.050	0.71^{NS}
Tri glyceride mg/dl	-0.018	0.89^{NS}
HDL mg/dl	0.182	0.18^{NS}
LDL mg/dl	-0.154	0.26^{NS}
VLDL mg/dl	-0.018	0.89^{NS}

- **R»** correlation coefficient. **+»** positive correlation. **-»** negative correlation. **S»** significant (p value <0.05). **NS»** insignificant (p value >0.05).

Table 5: Logistic regression on DVT risk factors

Variable	B	S.E.	Sig.	Exp(B)
Vitamin-D	0.017	0.068	0.80	1.017
Cholesterol	0.020	0.054	0.71	1.020
Triglyceride	0.041	0.074	0.58	1.041
HDL	-0.124	0.064	0.05	0.883
LDL	0.045	0.071	0.52	1.046
VLDL	-0.134	0.422	0.99	1.000

- B» Unstandardized regression coefficient S.E» Standard error
- Sig» Significance (p-value) Exp(B)» Exponentiated B (Odds ratio)

Discussion:

Venous thromboembolism (VTE) is a leading cause of maternal mortality, particularly in the UK, where it surpasses hemorrhage as the most common direct cause of death [6]. European studies estimate pregnancy-related VTE mortality at 8.5–14 per million live births [7]. It has been shown that 1,25-dihydroxyvitamin D, the active form of vitamin D, exerts anticoagulant effects in leukemia by upregulating thrombomodulin and downregulating tissue factor in monocytes and myelogenous cells. It has also been demonstrated that the vitamin D receptor may play an important role in thrombosis [8]. Moreover, previous data have shown that lipid-lowering drugs (statins, most notably rosuvastatin) are associated with a decreased risk of venous thrombosis, which may indicate a possible role for lipids in the pathophysiology of venous thrombosis [9]. Recent research has suggested that serum levels of vitamin D and lipids may serve as risk factors for DVT. The current study aims to evaluate the biochemical and clinical profiles of Sudanese women with deep vein thrombosis (DVT) compared to healthy pregnant and non-pregnant women. Vitamin D levels were markedly deficient in all three groups, with the lowest levels recorded among women with DVT and pregnant women. This deficiency may be exacerbated by increased melanin pigmentation and cultural practices limiting sun exposure, as outlined by Zittermann *et al*. (2021) and Sakem *et al*. (2022) [10,11]. Several studies, including those by Nassar *et al.* (2011) and Al-Shehri *et al.* (2020), have linked low maternal vitamin D levels with adverse pregnancy outcomes and increased thrombotic risk [12,13]. These findings are consistent with those of Wu *et al.* (2017) and Ameri *et al.* (2019), and further supported by a large population-based cohort study from 2022 involving over 18,000 individuals, which reported a stepwise increase in VTE risk with decreasing vitamin D levels [14,15]. However, systematic reviews such as that by Chilazi *et al.* (2022) and Mendelian randomization analyses by Manousaki *et al*. (2022) did not establish a direct causal relationship [16,17]. Nevertheless, the vitamin D receptor pathway may still be involved. Overall, these findings suggest that while vitamin D deficiency may not directly cause thrombosis, it likely reflects broader issues of systemic inflammation or health disparities, and remains a critical public health concern. Women with DVT and pregnant women exhibited significant lipid abnormalities, with increased levels of total cholesterol, triglycerides, LDL, and VLDL. Conversely, HDL was significantly reduced in the DVT group and displayed a borderline protective effect (OR 0.88; $p = 0.053$). These results align with the vasoprotective role of HDL described by Chen *et al.* (2021) and Barter *et al.* (2016), and with findings by Vaya *et al.* (2002) and Cushman *et al.* (2004), who observed links between dyslipidemia and thrombotic risk [18,19,20,21]. The decline in HDL may contribute to a pro-inflammatory, pro-coagulant environment, reinforcing its potential role in thrombus formation. These associations are consistently supported in the literature, with limited contradictory data. Clinical variables, including diabetes, prior thrombotic episodes, and the use of anticoagulant therapy, did not significantly affect most biochemical markers. However, vitamin D levels were lower among diabetic DVT patients and those with a history of thrombosis, potentially reflecting long-term metabolic or nutritional depletion, as also noted by Al-Mheid *et al.* (2012) and Lippi *et al.* (2013) [22,23]. Anticoagulant therapy had no significant effect on vitamin D or lipid profiles, which contrasts with findings from Western data indicating partial biomarker normalization following treatment (Di Nisio *et al.,* 2020; Roijer *et al.,* 2019) [24,25]. This suggests that anticoagulants may not sufficiently correct the prothrombotic biochemical state, thereby reinforcing the need for adjunctive therapies, such as vitamin D supplementation or novel antiplatelet agents. Among pregnant women, vitamin D levels declined progressively with increasing parity, supporting the concept of cumulative micronutrient depletion. This inverse relationship between gravida and vitamin D status aligns with reports by Bodnar *et al.* (2018), Nassar *et al.* (2011), and Al-Shehri *et al.* (2020), and suggests the need for enhanced nutritional screening and supplementation during pregnancy, particularly among high-parity women [12,13,26]. Age was not significantly correlated with any biomarker, challenging traditional age-related thrombosis models, as discussed by Kolluri *et al.* (2022) and Cushman *et al.* (2004) [27, 21]. Neither contraceptive use, nor delivery mode, significantly affected the studied biomarkers. The correlation between vitamin D levels and gravida remained the only consistently significant finding among the pregnancy subgroup. Binary logistic regression analysis

identified HDL as demonstrating a borderline protective effect (OR 0.88; p = 0.053), while other metabolic markers, including LDL, VLDL, cholesterol, triglycerides, and vitamin D, did not independently predict DVT risk (p > 0.05). These results highlight the need for population-specific guidelines in DVT risk assessment and management. Vitamin D deficiency is both widespread and clinically significant, warranting public health interventions. Anticoagulants appear insufficient to normalize the biochemical milieu associated with thrombosis, reinforcing the need for adjunct strategies such as vitamin D supplementation or antiplatelet therapy

Conclusion

This study highlights the significant prevalence of vitamin D deficiency and lipid abnormalities among Sudanese women with DVT, suggesting a potential contribution of these biochemical markers to thrombotic risk. While individual biomarkers such as HDL cholesterol demonstrated a borderline protective effect, the lack of strong predictive power from other markers indicates that DVT is a multifactorial condition influenced by a complex combination of genetic, environmental, and nutritional factors.

Recommendations

- Interventional studies are required to clarify temporal relationships between biomarker disturbances and DVT development.
- Direct laboratory measurements, for estimating LDL and VLDL levels, which may be more accurate and precision than Freidwald formula are required. Early screening for vitamin D and lipid levels in pregnant women and those at high risk should be study to help in early detection and prevention.
- Clinical trials should evaluate the benefits of vitamin D supplements or medications that reduce cholesterol in lowering the risk of DVT.
- More research is needed to study the interactions between vitamin D, genetic factors, and lifestyle for the prevention and management of thrombosis.

Study Limitations

Several limitations of this study should be acknowledged. First, the hospital-based sampling from two geographic regions (Khartoum and Northern State) may limit the external validity of the findings, particularly when extrapolating to rural populations or other Sudanese demographic groups. Second, although the total sample size (n = 165) was adequate for primary comparisons, it constrained more detailed subgroup analyses such as stratification based on diabetes severity or inherited thrombophilia markers. Third, several potential confounding variables were not measured, including dietary intake, accurate quantification of sun exposure, genetic variants (e.g., factor V Leiden), and inflammatory biomarkers (e.g., C-reactive protein), all of which may impact vitamin D status and lipid metabolism.

Authors' declaration:

We confirm that all the Tables in the manuscript belong to the current study. Authors sign on ethical considerations' Approval-Ethical Clearance: The project was approved by the local ethical committee in (Omdurman Islamic University's) Faculty of Medical Laboratory Science, in addition to ethical clearance from ministry of health in Sudan on (Jan. 2022).

Conflict of Interest: None

Funding: None

Author's contribution

Study conception & design: (Marwan A. Abdullah), Literature search: (Fayza A. Rahamtalla). Data acquisition: (Marwan A. Abdullah). Data analysis & interpretation: (Abdelmula M. Abdalla). Manuscript preparation: (Fayza A. Rahamtalla). Manuscript editing & review: (Abdelmula M. Abdalla).

References

1. Goldhaber SZ, Bounameaux H. Pulmonary embolism and deep vein thrombosis. Lancet. 2012;379(9828):1835–1846.
2. Judd SE, Tangpricha V. Vitamin D deficiency and risk for cardiovascular disease. Am J Med Sci. 2009;338(1):40–44.
3. Brøndum E, Borge G., Nordestgaard, Peter, Schnohr, Marianne Benn. Vitamin D and venous

thromboembolism: A prospective study. J Thromb Haemost. 2010;8(12):2686–2691.
4. Austin MA, Hokanson, J. E., & Edwards, K.L. Does hypertriglyceridemia cause cardiovascular disease? Circulation. 2005;112(16):2697–2708.
5. Ageno W, Becattini, C., Brighton, T., Selby, R., & Kamphuisen, P. Cardiovascular risk factors and venous thromboembolism: a prospective study. Thromb Haemost. 2008;99(6):1144–1148.
6. Lewis G. Why mothers die 2000–2002. The sixth report of the Confidential Enquiries into Maternal Deaths in the United Kingdom. London: RCOG Press; 2005.
7. Hogberg U, Lindmark G, Samuelsson S. Maternal mortality in Sweden, 1980–1988. Obstet Gynecol. 1994;84(2):240–244.
8. Wu-Wong JR, Nakane M, Ma J, Ruan X, Kroeger PE. Effects of vitamin D analogs on gene expression profiling in human coronary artery smooth muscle cells. Atherosclerosis. 2007;193(2):233–241.
9. Glynn,R. J., Danielson, E., Fonseca, F. A. H., Genest, J., Gotto, A. M., Kastelein, J. J. P., Koenig, W., Libby P., Lorenzatti, A. J., MacFadyen, J. G., Nordestgaard, B. G., Shepherd, J., Willerson, J.T., & Ridker, P. M. Randomized trial of rosuvastatin in the prevention of venous thromboembolism. New England Journal of Medicine. (2009) ; 360(18), 1851-1861.
10. Zittermann, A., Pilz, S., Hoffmann, H. and März, W. Vitamin D and mortality: Meta-analysis of individual participant data from a large consortium. J Clin Endocrinol Metab. 2021;106(10):e4108–e4119.
11. Sakem, B., Nock, C., Stanga, Z., Medina, P. and Christ-Crain, M . Impact of vitamin D deficiency on biochemical markers and clinical outcomes. Nutrients. 2022;14(5):925.
12. Nassar, N., Halligan, G. H., & Roberts, C. L . Hypovitaminosis D in pregnancy and its association with adverse pregnancy outcomes. Am J Perinatol. 2011;28(1):21–24.
13. Al-Shehri, M.A.Alzahrani, A.A., and Elnour, A. A. The relationship between vitamin D levels and thrombotic events in pregnant women. Int J Womens Health. 2020;12:43–50.
14. Wu J., He, Y., Zhang, L., & Zhang, L. Association between vitamin D and risk of venous thromboembolism: A meta-analysis. Thromb Res. 2017;157:38–44.
15. Ameri, A., Moradi, Y., Jafari, R., Moosazadeh, M. and Tabrizi, R . Vitamin D deficiency and thrombotic risk: A population-based study. J Thromb Haemost. 2019;17(3):378–384.
16. Chilazi, M., Choi, J., & Michos, E. D. Systematic review of vitamin D and venous thromboembolism. J Am Coll Cardiol. 2022;79(9):876–888.
17. Manousaki, D., Mitchell, R., Dudding, T., Haworth, S., Harroud, A., Forgetta, V. Mendelian randomization study of vitamin D and risk of thromboembolism. Lancet Haematol. 2022;9(2):e109–e118.
18. Chen, J., Kuang, J., Tang, X., Mao, L., Guo, X., Luo, G. and He, J. Protective role of HDL in vascular biology. Front Pharmacol. 2021;12:664183.
19. Barter, P., Gotto, A.M., LaRosa, J.C., Maroni, J., Szarek, M., Grundy, S.M. HDL cholesterol and cardiovascular risk. Nat Rev Cardiol. 2016;13(1):48–60.
20. Vaya, A., Mira, Y., Ferrando, F., Contreras, M.T., Estelles, A., España, F. & Aznar, J. Association between lipids and thrombosis. Thromb Res. 2002;107(1):31–35.
21. Cushman, M., Tsai, A.W., White, R.H., Heckbert, S.R., Rosamond, W.D., Enright, P. and Folsom, A.R. Lipoprotein levels and venous thrombosis risk. Blood. 2004;104(12):3933–3939.
22. Al Mheid, I., Patel, R.S., Tangpricha, V. and Quyyumi, A.A. Vitamin D deficiency and inflammation. Am J Cardiol. 2012;109(2):226–230.
23. Lippi G, et al. Vitamin D and thrombosis risk. Semin Thromb Hemost. 2013;39(3):273–281.
24. Di Nisio, M., van Es, N., Cesarman, G., Otten, H.M. and Büller, H.R. Effects of anticoagulants on lipid profiles. Thromb Haemost. 2020;120(4):613–620.
25. Roijer, A., Holmström, M., Odeberg, J. and Rånby, M. Vitamin D levels during anticoagulation therapy. Eur J Clin Invest. 2019;49(6):e13107.
26. Bodnar, L.M., Simhan, H.N., Catov, J.M., Roberts, J.M., Platt, R.W. and Diesel, J.C. High parity and vitamin D status. J Nutr. 2018;148(3):379–386.
27. Kolluri, R., Muddana, S.K. and Alikhan, R. Age-related changes in thrombotic risk. Vasc Med. 2022;27(3):235–243.