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Association of Vitamin D and Lipid Profile among Reproductive and Menopausal Groups of Women: A Cross-Sectional Study

Kongbrailatpam Jaishree Devi[1], Jaspreet Kaur[2], Bhumika Upadhyay[3]
[1](Department of Biochemistry, Hamdard Institute of Medical Sciences and Research, New Delhi, India)
[2](Department of Biochemistry, Hamdard Institute of Medical Sciences and Research, New Delhi, India)
[3](Department of Biochemistry, Hamdard Institute of Medical Sciences and Research, New Delhi, India)

Abstract:
Background*: Vitamin D is a lipid-soluble vitamin synthesized mainly under the skin during exposure to UVB (ultraviolet B) of sunlight. Due to the increasing prevalence of indoor lifestyles and various other metabolic and physiological factors, Vitamin D deficiency is now extremely widespread globally. Research has shown that Vitamin D deficiency (<20ng/ml) was found to be more prevalent in menopausal women than in reproductive age groups of women. This research aims to estimate serum vitamin D levels and lipid profiles in women of reproductive and menopausal age groups, and to investigate their metabolic associations.*
Materials and Methods*: This retrospective cross-sectional study was conducted at the HIMSR and HAHC Hospital, New Delhi. Two separate groups, consisting of 100 reproductive women (aged 18 - 40 years) and 100 menopausal women (aged>45 years and having attained menopause), were randomly selected. Serum vitamin D (25-hydroxyvitamin D) and lipid profile parameters (triglycerides, total cholesterol, HDL, non-HDL, LDL, and VLDL cholesterol levels) were analyzed from blood samples collected during their hospital visits from June 2024 to March 2025 using Abbott Architect i1000SR and Beckman Coulter AU480, respectively. Lastly, coefficients of Pearson's correlation and Levene's Independent sample T-tests were computed from the data collected using statistical software SPSS version 26.*
Results*: Results indicate that vitamin D was deficient in 50.5% of the overall study population. The mean vitamin D levels recorded were 22.19± 14.78 ng/ml and 21.97± 11.93 ng/ml in reproductive and menopausal women, respectively. Serum vitamin D in the overall study population shows significant negative correlation with Triglycerides (p=0.004) and VLDL cholesterol (p=0.019) levels, but significant positive correlation with HDL cholesterol (p=0.028). The t-test shows no significant differences between the respective mean parameters of the study groups.*
Conclusion: *This study establishes that vitamin D deficiency is highly prevalent and has a significant role in dyslipidaemia. Consequently, assessing vitamin D levels and providing supplementation to all women, irrespective of menstrual status, may be advantageous for the prevention and reversal of atherosclerosis and cardiovascular disorders.*

Keywords: *Atherosclerosis; Cardiovascular Disease; Dyslipidemia; Menopause; Vitamin D*

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I. Introduction

Vitamin D is a lipid-soluble vitamin synthesized mainly under the skin during exposure to UVB (ultraviolet B) of sunlight [1, 2]. It is a steroid hormone, which performs important roles in preserving calcium homeostasis, promoting bone health, and improving immune functions in our body [3, 4, 5]. Due to increasing indoor lifestyles and various other metabolic and physiological reasons, vitamin D deficiency has presently become highly prevalent worldwide [6, 7]. As per a recent study, vitamin D deficiency was found to be present in 88% of reproductive women in New Delhi. [8, 9]

Menopause is the cessation of the menstrual cycle for a minimum duration of at least 12 months caused by insufficient as well as declining secretions of estrogen and progesterone [10, 11]. Estrogen can also increase the kidneys' 1-alpha-hydroxylase activity, which activates vitamin D and further upregulates the vitamin D receptor (VDR). Therefore, the onset of menopause and subsequent drop in estrogen levels with ageing can cause decreased levels of serum vitamin D [12]. Few studies have further shown that a lack of vitamin D (< 20 ng/ml) was found to be more commonly seen in menopausal than in pre-menopausal age groups of women [13]. The term "dyslipidaemia" encompasses various types of imbalances in the levels of lipids found in the serum, like LDL-C (low-density

lipoprotein cholesterol), HDL-C (high-density lipoprotein cholesterol), TC (total cholesterol), and TG (triglycerides) [14].

Current literatures suggest that, at the molecular level, there is a close relationship between vitamin D as well as lipid metabolism, which hints towards a possible connection between vitamin D as well as lipid profile [15 - 18]. Various separate studies have also noted that both low levels of vitamin D and dyslipidaemia are linked to higher risks of atherosclerosis and cardiovascular conditions [19 - 24].

II. Aims And Objectives

This study aims to

- Evaluate the levels of serum vitamin D among reproductive and menopausal age groups of women,
- Evaluate the levels of serum lipid profile among reproductive and menopausal age groups of women, and
- Determine the metabolic associations of vitamin D with lipid profile.

III. Materials and Methods

Assuming 88% prevalence, with a 95% confidence level and 5% absolute precision, the actual sample size obtained after calculation was 162.27. However, it was increased, and the final total sample size of 200 was decided. A retrospective cross-sectional study design, in which the total sample size was further subdivided into two separate groups: "Reproductive group" consisting 100 reproductive women (18 - 40 years age) and "Menopausal group" consisting 100 menopausal women (age >45 years and had attained menopause) was finally planned and conducted at HIMSR and HAHC Hospital, New Delhi. Serum vitamin D (25-hydroxyvitamin D) and lipid profile parameters (which include triglycerides, total cholesterol, HDL, non-HDL, LDL, and VLDL cholesterol levels) were analyzed from the blood samples collected from randomly selected participants during their hospital visits from June 2024 to March 2025. Serum vitamin D is estimated using Abbott Architect i1000SR, which employs a chemiluminescent microparticle immunoassay (CMIA) to quantitatively determine 25-hydroxyvitamin D levels in human serum. Lipid profile parameters are measured using the Beckman Coulter AU480, which is based on spectrophotometry. Lastly, coefficients of Pearson's correlation and Levene's Independent sample T-tests were computed from the data collected using statistical software SPSS version 26. A value of $p < 0.05$ shall be considered significant.

Inclusion and exclusion criteria:

For the reproductive group, women aged 18 to 40 years who have a regular, normal menstrual cycle are included in the study. In addition, for the menopausal group, women aged> 45 years with at least 12 months of amenorrhea have been included.

Women with current pregnancy, menstrual disorders, bone fractures, hormonal therapy, oral contraceptive use, chronic smoking or alcoholic history, vitamin D supplementations or lipid-lowering agents, diabetes, hypertension, and other systemic diseases have been excluded from the study.

Statistical analysis

Table No. 1: Shows the cross-tabulation of Vitamin D statuses among the two study groups

Menstrual status	Vitamin D sufficient samples (%)	Vitamin D deficient samples (%)	Total sample
Reproductive group	46	54	100
Menopausal group	53	47	100
Total (%)	99 (= 45.5%)	101 (= 50.5%)	200 (100%)

Table No. 2: Shows group statistics of the mean parameters in the reproductive and menopausal groups of women

Parameter	Group	N (Sample)	Mean	Std. Deviation	Std. Error Mean
Age (years)	Reproductive	100	28.4800	6.32851	.63285
	Menopausal	100	57.0500	11.26752	1.12675
Vitamin- D (ng/ml)	Reproductive	100	22.1866	14.77492	1.47749
	Menopausal	100	21.9670	11.92609	1.19261

T. Cholesterol (mg/dl)	Reproductive	100	149.3810	44.36923	4.43692
	Menopausal	100	151.3130	56.88787	5.68879
Triglyceride (mg/dl)	Reproductive	100	146.4370	89.07812	8.90781
	Menopausal	100	158.1210	84.99626	8.49963
HDL-C (mg/dl)	Reproductive	100	38.4420	12.88310	1.28831
	Menopausal	100	38.9490	14.97281	1.49728
Non-HDL-C (mg/dl)	Reproductive	100	110.9390	37.62816	3.76282
	Menopausal	100	112.3640	48.65697	4.86570
LDL-C (mg/dl)	Reproductive	100	97.9330	32.46309	3.24631
	Menopausal	100	98.0528	38.00772	3.80077
VLDL-C (mg/dl)	Reproductive	100	30.3372	23.47657	2.34766
	Menopausal	100	32.5848	28.91781	2.89178

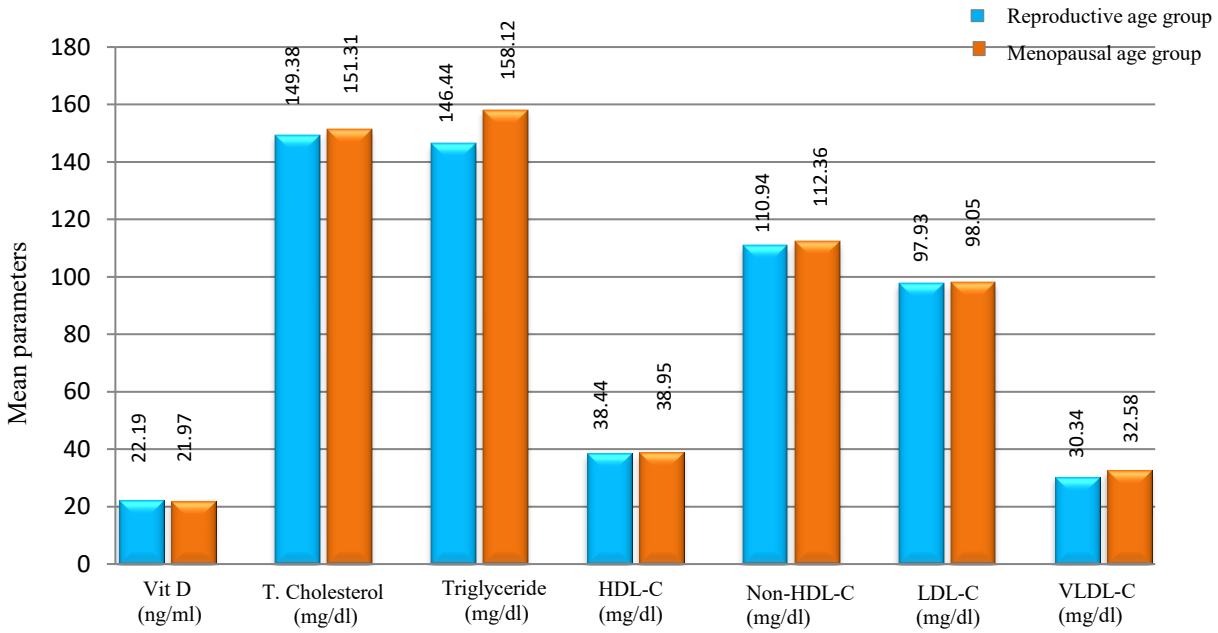

Fig. No 1: Shows bar chart comparison of mean parameters in Reproductive and Menopausal groups of women.

Table No. 3: Shows correlations of vitamin D levels with various parameters of lipid profiles.

		Age	Total Cholesterol	Triglycerides	HDL cholesterol	Non-HDL cholesterol	LDL cholesterol	VLDL cholesterol
Vitamin D (N = 200)	Correlation (R)	.008	-.014	-.201**	.156*	-.067	.007	-.166*
	Sig. (p value)	.916	.841	.004	.028	.348	.919	.019

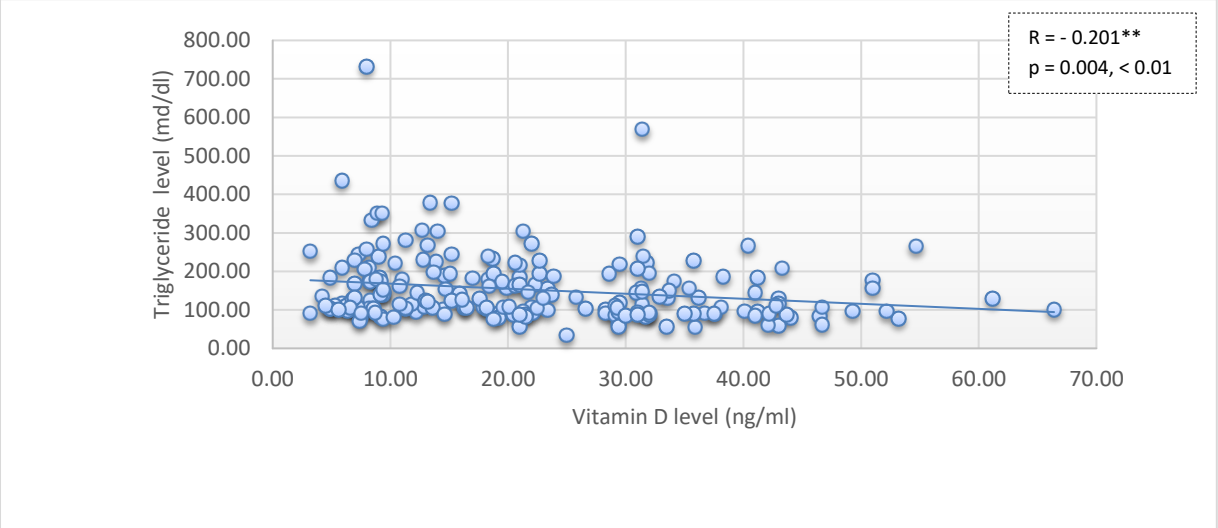

Fig. No. 2: Shows simple scatter plot of serum triglycerides vs vitamin D levels

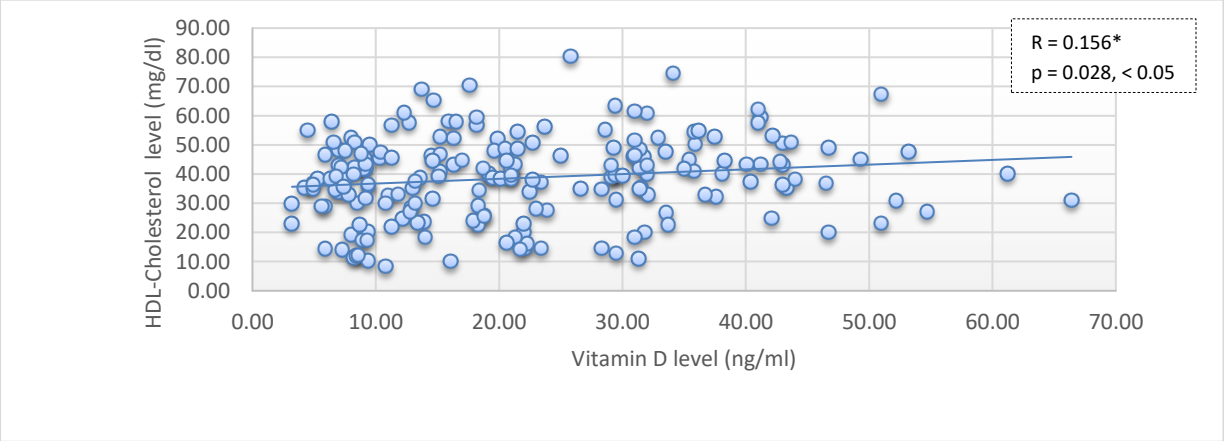

Fig. No. 3: Shows simple scatter plot of serum HDL cholesterol vs vitamin D levels

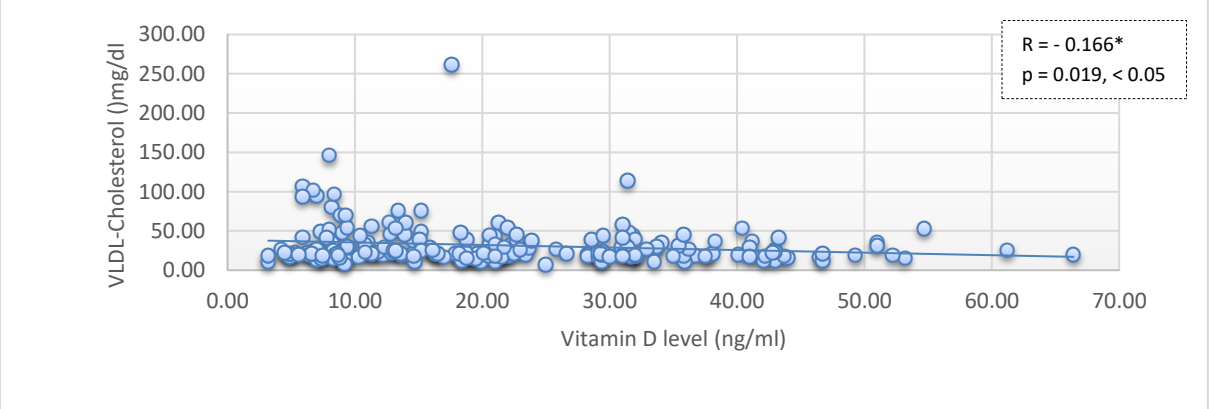

Fig. No. 4: Shows simple scatter plot of serum VLDL cholesterol vs Vitamin D levels

Table No. 4: Shows the findings of Levene's Independent Samples T-Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Age (years)	Equal variances assumed	40.773	.000	-22.108	198	.000	-28.57000	1.29231	-31.11846	-26.02154
	Equal variances not assumed.			-22.108	155.808	.000	-28.57000	1.29231	-31.12271	-26.01729
Vitamin D (ng/ml)	Equal variances assumed	7.070	.008	.116	198	.908	.21960.	1.89876	-3.52479	3.96399
	Equal variances not assumed.			.116	189.562	.908	.21960	1.89876	-3.52582	3.96502
T. cholesterol (mg/dl)	Equal variances assumed	2.517	.114	-.268	198	.789	-1.93200.	7.21447	-16.15906	12.29506
	Equal variances not assumed.			-.268	186.914	.789	-1.93200	7.21447	-16.16425	12.30025
Triglyceride (mg/dl)	Equal variances assumed	.106	.745	-.949	198	.344	-11.68400.	12.31230	-35.96407	12.59607
	Equal variances not assumed.			-.949	197.566	.344	-11.68400	12.31230	-35.96440	12.59640
HDL-Cholesterol (mg/dl)	Equal variances assumed	5.187	.024	-.257	198	.798	-.50700	1.97525	-4.40222.	3.38822
	Equal variances not assumed.			-.257	193.689	.798	-.50700	1.97525	-4.40275	3.38875
Non-HDL-Cholesterol (mg/dl)	Equal variances assumed	1.935	.166	-.232	198	.817	-1.42500	6.15092	-13.55472.	10.70472
	Equal variances not assumed.			-.232	186.219	.817	-1.42500	6.15092	-13.55944	10.70944
LDL-Cholesterol (mg/dl)	Equal variances assumed	2.106	.148	-.024	198	.981	-.11980.	4.99844	-9.97681	9.73721
	Equal variances not assumed.			-.024	193.273	.981	-.11980	4.99844	-9.97829	9.73869
VLDL-Cholesterol (mg/dl)	Equal variances assumed	.000	.986	-.603	198	.547	-2.24760	3.72477	-9.59291	5.09771
	Equal variances not assumed.			-.603	189.978	.547	-2.24760	3.72477	-9.59481	5.09961

IV. Results

The mean age obtained for the reproductive group is 28.48 ± 6.33 years, and that for the menopausal group is 57.05 ± 11.27 years (Table No. 2). Vitamin D deficiency showed a very high prevalence of 50.5% (Table No. 1) in the overall study population. While the mean vitamin D level observed was 22.19 ± 14.78 ng/ml in reproductive women and slightly decreased i.e., 21.97 ± 11.93 ng/ml in menopausal women (Table No. 2 and Fig. No 1), this difference was not statistically significant ($p > 0.05$) (Table No. 4). Further, Pearson's correlation studies (Table No. 3) showed that serum vitamin D has significant negative correlations with serum triglycerides ($R= -0.201$, $p = 0.004$) (Fig. No. 2) and VLDL cholesterol levels ($R= -0.166$, $p = 0.019$) (Fig. No. 4); but significant positive correlation with serum HDL cholesterol ($R= 0.156$, $p = 0.028$) (Fig. No. 3). Levene's T-test results showed no significant differences in the mean parameters (vitamin D and lipid profiles) obtained in this study (Table No. 4).

V. Discussion

The association between vitamin D deficiency and dyslipidaemia, which further leads to atherosclerosis, is complex and involves multiple mechanisms. Mechanisms proposed by various studies are being discussed as follows: a) **Foam cell formation**: Vitamin D regulates lipid metabolism and can influence foam cell formation. Vitamin D deficiency may contribute to increased lipid uptake and accumulation in macrophages, leading to foam cell formation and atherosclerosis. b) **Inflammation and oxidative stress**: Vitamin D has anti-inflammatory and antioxidant properties. Deficiency can lead to increased inflammation and oxidative stress, promoting atherosclerotic plaque formation. c) **Endothelial dysfunction**: Vitamin D receptors (VDRs) are present in endothelial cells. Vitamin D deficiency can lead to impaired endothelial function, increased vascular stiffness, and reduced nitric oxide production. d) **Renin-angiotensin-aldosterone system (RAAS) activation**: Vitamin D deficiency can activate the RAAS, leading to increased blood pressure, vascular remodelling, and atherosclerosis. e) **Impaired vascular smooth muscle cell function**: Vitamin D deficiency can affect vascular smooth muscle cell function, leading to increased vascular stiffness and atherosclerosis. f) **Epigenetic modifications and gene regulation**: Vitamin D deficiency can influence epigenetic modifications and gene regulation, affecting the

expression of genes involved in atherosclerosis. In spite of the smaller sample size, our study findings reflect a close molecular link between lipid metabolism and vitamin D, which is in alignment with the current literature.[21-25]

VI. Conclusion

This study establishes that vitamin D deficiency is highly prevalent and has a significant role in dyslipidaemia. Consequently, assessing vitamin D levels and providing supplementation to all women, irrespective of menstrual status, may be advantageous for the prevention and reversal of atherosclerosis and cardiovascular disorders.

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