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Evaluation of Vitamin D Deficiency and Its Association with Lipid Profile and Inflammatory Biomarkers in Coronary Artery Disease Patients.

Amit Prakash Darira*, and Jenny Koshy.

General Medicine Department, New Thergaon Hospital, Thergaon, Pune, Pimpri-Chinchwad, Maharashtra 411033, India.

ABSTRACT

Vitamin D deficiency is increasingly recognized as a potential non-traditional risk factor in the pathogenesis of coronary artery disease (CAD). It may influence cardiovascular outcomes by modulating lipid metabolism and systemic inflammation. This study aimed to evaluate the prevalence of vitamin D deficiency and its association with lipid profile and inflammatory biomarkers in CAD patients. A cross-sectional observational study was conducted over one year among 40 patients with angiographically confirmed CAD. Serum 25-hydroxy-vitamin D [25(OH)D] levels were measured and categorized as deficient, insufficient, or sufficient. Fasting lipid profile, high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and tumour necrosis factor-α (TNF-α) levels were assessed. Statistical analysis was performed using SPSS version 26, with significance set at $p < 0.05$. Vitamin D deficiency (<20 ng/mL) was found in 55% of patients. Deficient individuals had significantly higher total cholesterol, LDL-C, and lower HDL-C compared to sufficient individuals. Inflammatory biomarkers (hs-CRP, IL-6, TNF-α) were significantly elevated in the deficient group. An inverse correlation was observed between vitamin D levels and both lipid abnormalities and inflammatory markers. Vitamin D deficiency is common in CAD patients and is associated with dyslipidemia and elevated inflammatory biomarkers. Monitoring and correcting vitamin D deficiency may be beneficial in comprehensive cardiovascular risk management.

Keywords: Vitamin D deficiency, Coronary artery disease, Inflammatory biomarkers

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Corresponding author

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INTRODUCTION

Coronary artery disease (CAD) remains a leading cause of morbidity and mortality globally, with a complex etiology involving both traditional and emerging risk factors. Among the non-traditional contributors, Vitamin D deficiency has garnered increasing attention due to its potential role in cardiovascular health. Vitamin D, a fat-soluble secosteroid hormone, is well-known for its regulatory function in calcium and bone metabolism. However, recent evidence suggests it may exert immunomodulatory, anti-inflammatory, and cardioprotective effects. Hypovitaminosis D has been associated with endothelial dysfunction, increased arterial stiffness, and heightened inflammatory response—factors that contribute significantly to the pathogenesis and progression of atherosclerosis.

Lipid abnormalities and systemic inflammation are central to the development of CAD. Elevated low-density lipoprotein (LDL), reduced high-density lipoprotein (HDL), and increased levels of inflammatory markers such as C-reactive protein (CRP) have consistently been linked to adverse cardiovascular outcomes. Studies indicate that Vitamin D may influence lipid metabolism and modulate inflammatory pathways, thereby affecting cardiovascular risk. However, the precise relationship between Vitamin D status, lipid profile, and inflammatory markers in CAD patients remains under-explored, particularly in the Indian population [1-4].

This study aims to evaluate the prevalence of Vitamin D deficiency in CAD patients and investigate its association with lipid parameters and inflammatory biomarkers to better understand its role in cardiovascular risk stratification and management.

METHODOLOGY

The present cross-sectional, observational study was carried out in the Department of Cardiology of a tertiary-care teaching hospital over a period of one year (January 2024 – December 2024). Consecutive adults (≥ 30 years) with angiographically confirmed coronary artery disease who attended the outpatient clinic or were admitted for elective interventions were screened for eligibility. A total of 40 patients fulfilled the inclusion criteria and provided written informed consent; the institutional ethics committee had approved the protocol before enrolment.

Patients with chronic kidney disease (stage ≥ 3), chronic liver disease, malabsorption syndromes, active infection, autoimmune disorders, malignancy, or current use of vitamin D or lipid-lowering supplements were excluded to minimise confounding. Baseline demographic data, cardiovascular risk factors, medication history, and anthropometric measurements were recorded using a pre-validated case-record form. Clinical severity of CAD was documented from coronary angiography reports, and the SYNTAX score was calculated for each participant.

After an overnight fast of 10–12 hours, venous blood samples were obtained between 8 am and 10 am. Serum 25-hydroxy-vitamin D [25(OH)D] concentrations were measured by chemiluminescent immunoassay, and vitamin D status was categorised as deficient (< 20 ng/mL), insufficient (20–29 ng/mL), or sufficient (≥ 30 ng/mL). The lipid profile—including total cholesterol, LDL-cholesterol, HDL-cholesterol, and triglycerides—was analysed enzymatically on an automated analyser. High-sensitivity C-reactive protein (hs-CRP) was quantified by nephelometry, while interleukin-6 (IL-6) and tumour necrosis factor-α (TNF-α) were assessed using commercially available ELISA kits, strictly adhering to manufacturers' instructions.

Data were entered into Microsoft Excel and analysed with SPSS version 26.0. Continuous variables were expressed as mean ± standard deviation or median (inter-quartile range) depending on normality, and categorical variables as frequencies and percentages. Comparisons of lipid parameters and inflammatory biomarkers across vitamin D status groups were performed using one-way ANOVA or the Kruskal–Wallis test for continuous data and the chi-square test for categorical data. Spearman's rank correlation coefficient evaluated the relationship between serum 25(OH)D levels and biochemical markers, while multivariable linear regression identified independent predictors after adjusting for age, sex, BMI, diabetes, hypertension, and statin use. A p-value < 0.05 was considered statistically significant.

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RESULTS

Table 1: Distribution of Vitamin D Status and Associated Lipid Profile (n = 40)

Parameter	Vitamin D Deficient (<20 ng/mL) (n=22)	Vitamin D Insufficient (20–29 ng/mL) (n=10)	Vitamin D Sufficient (≥30 ng/mL) (n=8)	*p*-value
Total Cholesterol (mg/dL)	210.5 ± 32.4	192.7 ± 28.9	178.3 ± 25.7	0.018*
LDL-C (mg/dL)	136.2 ± 26.1	120.6 ± 20.8	112.4 ± 19.5	0.021*
HDL-C (mg/dL)	34.1 ± 6.8	39.3 ± 5.4	42.7 ± 6.1	0.007*
Triglycerides (mg/dL)	168.4 ± 41.6	154.2 ± 36.3	148.7 ± 32.8	0.196

*Statistically significant at $p < 0.05$

Table 2: Inflammatory Biomarkers According to Vitamin D Status

Biomarker	Vitamin D Deficient (<20 ng/mL) (n=22)	Vitamin D Insufficient (20–29 ng/mL) (n=10)	Vitamin D Sufficient (≥30 ng/mL) (n=8)	*p*-value
hs-CRP (mg/L)	4.8 ± 1.6	3.5 ± 1.2	2.9 ± 1.1	0.011*
IL-6 (pg/mL)	7.3 ± 2.4	5.6 ± 1.7	4.1 ± 1.3	0.004*
TNF-α (pg/mL)	6.1 ± 1.9	5.0 ± 1.5	3.7 ± 1.4	0.010*

*Statistically significant at $p < 0.05$

DISCUSSION

The present study investigated the prevalence of vitamin D deficiency and its association with lipid abnormalities and inflammatory biomarkers in patients with angiographically proven coronary artery disease (CAD). A striking 55 % of participants exhibited frank vitamin D deficiency, while only 20 % were vitamin-D-replete, underscoring how common hypovitaminosis D remains even in a sun-rich region. This prevalence aligns with earlier Indian cohorts and mirrors global reports that place vitamin D deficiency in 40–70 % of cardiac populations, highlighting a consistent yet under-addressed cardiovascular risk milieu.

The most salient finding was the graded, inverse relationship between serum 25-hydroxy-vitamin D [25(OH)D] concentrations and atherogenic lipid fractions. Total cholesterol and LDL-cholesterol differed by roughly 30 mg/dL between deficient and sufficient groups, and HDL-cholesterol rose by nearly 9 mg/dL across the same gradient. These trends persisted after adjusting for potential confounders such as diabetes, body-mass index, and statin use (data not shown). Mechanistically, vitamin D is believed to modulate hepatic very-low-density lipoprotein synthesis via down-regulation of sterol regulatory element-binding protein-1c and to enhance reverse cholesterol transport by up-regulating apolipoprotein AI. The favourable HDL increment observed here lends indirect support to this pathway. While the triglyceride reduction did not reach statistical significance, its numerical fall in higher vitamin-D strata suggests that a larger sample might detect a meaningful difference.

Equally important is the clear association between vitamin D status and systemic inflammation. High-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6) and tumour necrosis factor-α (TNF-α) all demonstrated a stepwise decrease with rising 25(OH)D concentrations, with mean IL-6 levels almost halved in vitamin-D-sufficient patients. Vitamin D's immunomodulatory role involves inhibition of nuclear factor-κB activation, promotion of anti-inflammatory cytokines, and suppression of pro-inflammatory Th1 responses. Given that chronic, low-grade inflammation propels every stage of atherogenesis—from endothelial dysfunction to plaque rupture—the observed biomarker profile offers a plausible biological link between hypovitaminosis D and CAD progression.

Our findings have several clinical implications. First, routine screening for vitamin D deficiency in CAD patients might identify a modifiable, non-traditional risk factor. Second, correcting deficiency could synergise with lipid-lowering and anti-inflammatory strategies, although interventional trials are needed. Randomised supplementation studies have produced mixed cardiovascular results, often owing to

heterogeneous populations, dosing regimens and endpoints. Nevertheless, the consistent biochemical associations demonstrated here provide a rational basis for well-designed, adequately powered outcome trials in high-risk groups.

The study has limitations. Its cross-sectional design precludes causal inference; low vitamin D levels may simply mark, rather than mediate, poorer cardiometabolic health. The modest sample size (n = 40) limits generalisability and may have reduced statistical power for triglyceride differences. Seasonal variability in sunlight exposure was not explicitly captured, although blood sampling windows were standardised to mornings to mitigate diurnal fluctuation. Finally, we did not measure parathyroid hormone or fibroblast growth factor-23, both of which interact with vitamin D metabolism and cardiovascular physiology.

These constraints, the present work adds to a growing body of evidence linking vitamin D deficiency to an adverse biochemical milieu in CAD. Future longitudinal studies should explore whether sustained repletion favourably alters lipid and inflammatory profiles and, ultimately, clinical outcomes such as myocardial infarction and heart-failure progression. Until such data emerge, clinicians could consider vitamin D status a useful adjunct in the comprehensive cardiovascular risk assessment of CAD patients [5-10].

CONCLUSION

Vitamin D deficiency is common in CAD patients and is associated with dyslipidemia and elevated inflammatory biomarkers. Monitoring and correcting vitamin D deficiency may be beneficial in comprehensive cardiovascular risk management.

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