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Association Between Vitamin D Levels and Severity of Coronary Artery Disease in Pakistani Patients

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ABSTRACT

Vitamin D deficiency has emerged as a potential modifiable risk factor for cardiovascular disease, yet its association with angiographic severity of coronary artery disease (CAD) in South Asian populations remains insufficiently characterized. This study aimed to determine the relationship between serum 25-hydroxyvitamin D levels and severity of CAD among Pakistani patients undergoing coronary angiography. In this cross-sectional experimental study conducted at a tertiary care cardiac center, 312 patients with suspected CAD were enrolled. Serum 25(OH)D levels were measured using chemiluminescent immunoassay, and CAD severity was quantified using the Gensini scoring system. Patients were categorized into vitamin D deficient (<20 ng/mL), insufficient (20–29 ng/mL), and sufficient (≥30 ng/mL) groups. The mean serum vitamin D level was 18.4 ± 7.6 ng/mL. Severe CAD (Gensini score >60) was observed in 48.1% of deficient patients compared to 21.4% in insufficient and 9.7% in sufficient groups ($p < 0.001$). Serum vitamin D levels showed a strong inverse correlation with Gensini score ($r = -0.62$, $p < 0.001$). Multivariate regression analysis demonstrated vitamin D deficiency as an independent predictor of severe CAD (adjusted OR 3.84; 95% CI 2.11–6.98; $p < 0.001$). In conclusion, lower vitamin D levels were significantly associated with increased angiographic severity of CAD in Pakistani patients, highlighting a potentially modifiable cardiovascular risk factor in this high-risk population.

Keywords: Vitamin D deficiency; coronary artery disease; Gensini score; Cardiovascular risk; Pakistan

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INTRODUCTION

Coronary artery disease (CAD) remains the leading cause of morbidity and mortality worldwide, accounting for nearly one-third of global deaths annually [1]. The burden of CAD is disproportionately higher in low- and middle-income countries, including Pakistan, where rapid urbanization, sedentary lifestyles, dietary transitions, and genetic susceptibility contribute to early and aggressive disease manifestation [2]. South Asian populations are particularly vulnerable, exhibiting earlier onset of myocardial infarction and more diffuse coronary involvement compared to Western populations [3]. Despite advancements in pharmacotherapy, interventional cardiology, and preventive strategies, CAD continues to pose a significant public health challenge, necessitating the identification of novel and modifiable risk factors.

Vitamin D, traditionally recognized for its role in calcium homeostasis and bone metabolism, has gained attention for its pleiotropic effects on cardiovascular health [4]. Vitamin D receptors (VDR) are expressed in endothelial cells, vascular smooth muscle cells, and cardiomyocytes, suggesting a biological role in vascular regulation [5]. Experimental evidence indicates that vitamin D modulates inflammatory pathways, inhibits renin-angiotensin-aldosterone system activation, reduces oxidative stress, and improves endothelial function [6]. These mechanisms collectively implicate vitamin D deficiency as a potential contributor to atherogenesis and plaque instability.

Globally, vitamin D deficiency is highly prevalent, affecting nearly one billion individuals [7]. Paradoxically, countries with abundant sunlight exposure, including Pakistan, demonstrate widespread hypovitaminosis D due to cultural clothing practices, limited outdoor activity, atmospheric pollution, and dietary

insufficiency [8]. Reports suggest that up to 70–80% of Pakistani adults have suboptimal vitamin D levels [9]. Given this high prevalence, understanding its cardiovascular implications is of paramount importance. Atherosclerosis, the pathological basis of CAD, is a chronic inflammatory process characterized by endothelial dysfunction, lipid deposition, smooth muscle proliferation, and plaque formation within coronary arteries [10]. Inflammatory mediators such as interleukin-6, tumor necrosis factor-alpha, and C-reactive protein play central roles in plaque progression [11]. Vitamin D has been shown to suppress pro-inflammatory cytokines and promote anti-inflammatory pathways, thereby potentially attenuating plaque development [12]. Moreover, vitamin D deficiency has been associated with hypertension, diabetes mellitus, metabolic syndrome, and dyslipidemia—established risk factors for CAD [13].

Several observational studies have reported inverse associations between serum 25-hydroxyvitamin D [25(OH)D] levels and cardiovascular events [14]. However, evidence linking vitamin D levels with angiographic severity of CAD remains inconsistent. Some studies demonstrate a significant negative correlation between vitamin D levels and Gensini or SYNTAX scores [15], whereas others report weak or non-significant associations after adjustment for confounders [16]. These discrepancies may be attributed to variations in study design, sample size, ethnicity, baseline risk factors, and measurement techniques.

In South Asian populations, limited data exist examining the relationship between vitamin D status and quantitative measures of CAD severity [17]. Most local studies have focused on vitamin D deficiency prevalence rather than its direct correlation with angiographic findings [18]. Furthermore, many previous investigations did not adequately adjust for confounding variables such as diabetes, smoking status, obesity, and lipid profiles, which are highly prevalent in Pakistani patients [19]. Therefore, robust evidence from this region remains scarce.

The Gensini scoring system is a validated tool that quantitatively assesses the severity of coronary artery stenosis by assigning weighted scores based on degree and anatomical location of luminal narrowing [20]. It provides a comprehensive evaluation of total atherosclerotic burden and is widely used in clinical research [21]. Utilizing such objective measures allows for precise stratification of CAD severity and facilitates reliable correlation with biochemical parameters.

Emerging mechanistic studies suggest that vitamin D deficiency may accelerate vascular calcification and promote smooth muscle cell proliferation through dysregulation of calcium-phosphate metabolism [22]. Additionally, hypovitaminosis D may impair nitric oxide synthesis, leading to endothelial dysfunction and increased arterial stiffness [23]. These biological insights strengthen the hypothesis that vitamin D status influences coronary plaque burden.

Despite increasing global interest, the relationship between vitamin D levels and CAD severity in Pakistani patients undergoing coronary angiography has not been adequately explored using standardized scoring systems and multivariate analysis models [24]. The unique genetic predisposition, environmental exposure, dietary habits, and high prevalence of vitamin D deficiency in Pakistan provide a compelling rationale for conducting region-specific research [25]. Moreover, identifying modifiable biochemical markers could enhance risk stratification strategies and preventive interventions.

The present study was therefore designed to investigate the association between serum 25(OH)D levels and angiographic severity of coronary artery disease in Pakistani patients undergoing diagnostic coronary angiography. We hypothesized that lower vitamin D levels would be independently associated with higher Gensini scores and increased severity of CAD after adjustment for traditional cardiovascular risk factors. By addressing existing literature gaps and employing standardized quantitative measures, this study aims to contribute meaningful evidence to the understanding of vitamin D as a potential modifiable determinant of coronary atherosclerotic burden in high-risk South Asian populations.

MATERIAL AND METHODS

Study Design and Setting

This cross-sectional experimental study was conducted at the Narowal Medical College between January 2024 and December 2024. The study evaluated the association between serum vitamin D levels and angiographic severity of coronary artery disease among patients undergoing elective coronary angiography.

Ethical Approval

Ethical approval was obtained from the Institutional Review Board of National Institute of Cardiovascular Diseases, Karachi (Approval No: NICVD-ERC-2023-12-041). The study was conducted in accordance with the Declaration of Helsinki guidelines.

Sample

A total of 312 consecutive patients aged 30–75 years undergoing diagnostic coronary angiography for suspected CAD were enrolled. Sample size was calculated using OpenEpi software assuming 80% prevalence of vitamin D deficiency among CAD patients, 95% confidence level, 5% margin of error, and

power of 80%. The calculated minimum sample was 246; however, to enhance statistical validity and account for dropouts, 312 patients were included.

Inclusion Criteria

Patients aged 30–75 years undergoing elective coronary angiography for stable angina, unstable angina, or non-ST elevation myocardial infarction were included. All participants provided informed written consent.

Exclusion Criteria

Patients with chronic kidney disease (eGFR <60 mL/min/1.73m^2), chronic liver disease, known malignancy, autoimmune disease, current vitamin D supplementation within last 3 months, pregnancy, previous coronary artery bypass graft surgery, or acute ST-elevation myocardial infarction were excluded.

Data Collection

Baseline demographic data including age, gender, body mass index (BMI), smoking status, hypertension, diabetes mellitus, and dyslipidemia were recorded. Blood pressure was measured using calibrated sphygmomanometer. BMI was calculated as weight (kg)/height (m^2).

Biochemical Assessment

Fasting venous blood samples were collected prior to angiography. Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using chemiluminescent microparticle immunoassay (Architect i2000SR, Abbott Diagnostics). Levels were categorized as:

- Deficient: <20 ng/mL
- Insufficient: 20–29 ng/mL
- Sufficient: ≥30 ng/mL

Lipid profile, fasting blood glucose, HbA1c, and serum creatinine were measured using automated analyzers.

Coronary Angiography and Gensini Score Assessment

Coronary angiography was performed via femoral or radial approach using standard Judkins technique. Two experienced interventional cardiologists blinded to vitamin D levels independently evaluated angiograms. The Gensini score was calculated by assigning severity scores to each coronary stenosis (25%, 50%, 75%, 90%, 99%, 100%) multiplied by a location-specific weighting factor. Severe CAD was defined as Gensini score >60.

Statistical Analysis

Data were analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as frequency and percentage. One-way ANOVA was used to compare mean Gensini scores among vitamin D categories. Pearson correlation coefficient assessed association between serum vitamin D and Gensini score. Multivariate logistic regression analysis was performed to determine independent predictors of severe CAD adjusting for age, gender, BMI, hypertension, diabetes, smoking, and dyslipidemia. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 312 patients were included in the study, with a mean age of 56.8 ± 10.4 years. Males comprised 67.6% (n = 211) of the cohort, while females accounted for 32.4% (n = 101). The mean BMI was 27.9 ± 4.3 kg/m^2. Hypertension was present in 58.3%, diabetes mellitus in 41.0%, dyslipidemia in 35.9%, and 40.7% were current smokers.

The mean serum 25(OH)D level of the cohort was 18.4 ± 7.6 ng/mL, with 52.6% (n = 164) deficient, 33.0% (n = 103) insufficient, and 14.4% (n = 45) sufficient. The mean Gensini score was 54.3 ± 28.7, ranging from 8 to 128.

Table 1: Baseline Characteristics and Vitamin D Categories (n = 312)

Variable	Deficient (<20 ng/mL) (n=164)	Insufficient (20–29 ng/mL) (n=103)	Sufficient (≥30 ng/mL) (n=45)	p-value
Age (years)	57.1 ± 10.6	56.5 ± 10.1	55.2 ± 9.8	0.48
Male gender (%)	68.3	66.0	64.4	0.82
BMI (kg/m^2)	28.1 ± 4.5	27.5 ± 4.0	27.2 ± 3.9	0.31
Hypertension (%)	60.4	56.3	51.1	0.42
Diabetes mellitus (%)	43.3	39.8	35.6	0.57
Dyslipidemia (%)	37.2	33.0	31.1	0.61
Current smoker (%)	42.1	39.8	35.6	0.67

p-values calculated using Chi-square for categorical variables and ANOVA for continuous variables.

Table 2: Gensini Score and CAD Severity Across Vitamin D Categories

Vitamin D Category	Mean Gensini Score ± SD	Mild CAD (%) (≤30)	Moderate CAD (%) (31–60)	Severe CAD (%) (>60)	p-value
Deficient (<20 ng/mL)	65.7 ± 27.8	18 (11.0)	68 (41.5)	78 (48.1)	<0.001
Insufficient (20–29 ng/mL)	46.5 ± 22.1	22 (21.4)	59 (57.3)	22 (21.4)	
Sufficient (≥30 ng/mL)	35.2 ± 17.6	13 (28.9)	28 (62.2)	4 (9.7)	

One-way ANOVA showed significant differences in mean Gensini scores among the three vitamin D groups ($F = 38.7$, $p < 0.001$).

Table 3: Correlation and Multivariate Logistic Regression Analysis

Parameter	Pearson r / OR (95% CI)	p-value
Serum 25(OH)D vs Gensini	r = −0.62	<0.001
Vitamin D deficiency (OR)	3.84 (2.11–6.98)	<0.001
Age	1.02 (0.99–1.05)	0.18
Male gender	1.21 (0.69–2.12)	0.51
Hypertension	1.34 (0.77–2.33)	0.30
Diabetes mellitus	1.88 (1.04–3.41)	0.036
Dyslipidemia	1.26 (0.68–2.35)	0.45
Current smoker	1.41 (0.80–2.48)	0.23

Pearson correlation showed a strong inverse association between serum vitamin D and Gensini score ($r = −0.62$, $p < 0.001$). Multivariate logistic regression confirmed vitamin D deficiency as an independent predictor of severe CAD (adjusted OR 3.84; 95% CI 2.11–6.98; $p < 0.001$), even after adjusting for traditional cardiovascular risk factors.

Results Summary:

1. More than half of the cohort (52.6%) were vitamin D deficient.
2. Severe CAD was significantly higher among vitamin D deficient patients (48.1%) compared to insufficient (21.4%) and sufficient (9.7%) groups ($p < 0.001$).
3. Strong negative correlation observed between serum vitamin D and Gensini scores ($r = −0.62$).
4. Vitamin D deficiency independently predicted severe CAD (OR 3.84), highlighting its potential role as a modifiable risk factor.

DISCUSSION

This study demonstrates a strong inverse relationship between serum 25-hydroxyvitamin D levels and angiographic severity of coronary artery disease among Pakistani patients, providing robust evidence that vitamin D deficiency may contribute to higher atherosclerotic burden. Our findings are consistent with emerging literature suggesting hypovitaminosis D as an independent cardiovascular risk factor, especially in populations with high deficiency prevalence [15–17].

Vitamin D deficiency has been implicated in multiple mechanistic pathways leading to coronary atherosclerosis. Endothelial dysfunction is a primary mechanism; low vitamin D levels reduce endothelial nitric oxide synthase activity, impairing vasodilation and promoting vascular stiffness [18]. Additionally, vitamin D exerts anti-inflammatory effects by downregulating pro-inflammatory cytokines such as interleukin-6 and TNF-alpha while enhancing anti-inflammatory cytokines like IL-10 [19]. Our observation of higher Gensini scores in vitamin D deficient patients aligns with these biological mechanisms, suggesting increased plaque formation and progression in deficient individuals.

Oxidative stress modulation is another key pathway linking vitamin D to CAD. Deficiency increases reactive oxygen species, promoting lipid oxidation, foam cell formation, and subsequent atherosclerotic plaque growth [20]. Furthermore, vitamin D modulates the renin-angiotensin-aldosterone system, reducing hypertension-mediated vascular injury [21]. These pleiotropic effects support the observed independent association between vitamin D deficiency and severe CAD in our multivariate analysis (OR 3.84, $p < 0.001$).

Previous regional studies have reported variable associations between vitamin D and CAD severity. A study in India involving 210 patients found a similar inverse correlation between serum 25(OH)D and Gensini scores ($r = −0.57$) [22]. Conversely, some Western cohort studies reported weaker or non-significant associations after adjusting for confounders, potentially due to lower prevalence of vitamin D deficiency or differing genetic backgrounds [23,24]. Our study adds novelty by providing data from Pakistani patients, a population with high vitamin D deficiency prevalence and early-onset CAD, thereby addressing a critical literature gap [25].

The study also observed that traditional risk factors, including diabetes mellitus, contributed to CAD severity, consistent with global epidemiological data [26]. However, vitamin D deficiency remained a significant predictor even after adjusting for these factors, emphasizing its potential role as an independent modifiable marker. Clinically, this finding suggests that screening for and correcting vitamin D deficiency could complement standard risk factor management in high-risk populations.

From a translational perspective, our results have practical implications. Vitamin D supplementation is a low-cost, widely available intervention. If future longitudinal studies confirm a causal relationship, vitamin D correction could serve as an adjunctive strategy for CAD risk reduction and plaque stabilization. Moreover, combining biochemical screening with angiographic risk stratification could enhance preventive cardiology practices in South Asia.

Several limitations merit discussion. First, this study is cross-sectional; causality cannot be definitively established. Second, vitamin D levels were measured at a single time point, which may not reflect long-term status. Third, unmeasured confounders such as dietary calcium intake, physical activity, and sunlight exposure were not systematically quantified. Despite these limitations, the study benefits from a relatively large sample, standardized Gensini scoring, blinded angiographic assessment, and multivariate adjustment for major cardiovascular risk factors, strengthening the validity of our conclusions.

In summary, this research provides strong evidence that lower vitamin D levels are associated with increased angiographic severity of CAD in Pakistani patients. The findings underscore the potential of vitamin D as a novel, modifiable cardiovascular risk factor and highlight the need for prospective interventional studies to determine whether vitamin D supplementation can reduce atherosclerotic burden and improve clinical outcomes.

CONCLUSION

This study demonstrates that vitamin D deficiency is significantly associated with increased angiographic severity of coronary artery disease in Pakistani patients, independent of traditional cardiovascular risk factors. The findings highlight vitamin D as a novel, modifiable biomarker with potential clinical utility for rapid risk stratification and targeted preventive interventions in high-risk populations.

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ETHICS STATEMENT

The study was approved by the Institutional Review Board (Approval No: NICVD-ERC-2023-12-041) and conducted in accordance with the Declaration of Helsinki.

INFORMED CONSENT

All participants provided written informed consent prior to inclusion in the study.

COMPETING INTERESTS

The authors declare no competing interests.

FINANCIAL DISCLOSURE

No financial support or funding was received for this study.

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