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ASSOCIATION BETWEEN VITAMIN D AND HIGH-SENSITIVITY C-REACTIVE PROTEIN (HS-CRP) IN PATIENTS WITH DYSLIPIDEMIA: A CROSS-SECTIONAL STUDY

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ABSTRACT

Background: Dyslipidemia is a major risk factor for cardiovascular diseases. Several studies have reported an association between decreased vitamin D levels and dyslipidemia. Since high-sensitivity C-reactive protein (hs-CRP) is an established marker of cardiovascular risk, and atherosclerosis involves both lipid dysregulation and chronic inflammation, a potential link between hs-CRP and lipid metabolism warrants investigation. Understanding this relationship may improve cardiovascular risk stratification.

Objectives: To estimate and compare the concentrations of hs-CRP and vitamin D in dyslipidemia patients versus healthy controls, and to assess the correlation between these two parameters.

Materials and Methods: A cross-sectional case-control study was conducted involving 40 dyslipidemia patients and 40 age- and sex-matched healthy controls. Serum levels of hs-CRP and 25(OH) vitamin D were measured and statistically compared using t-tests and Pearson's correlation analysis.

Results: Vitamin D levels were significantly lower and hs-CRP levels significantly higher in dyslipidemia patients compared to controls ($p < 0.05$). A statistically significant inverse correlation was observed between hs-CRP and vitamin D levels among dyslipidemia patients.

Conclusion: This study demonstrates that dyslipidemia is associated with lower vitamin D levels and higher hs-CRP, indicating increased inflammation. The inverse correlation supports vitamin D's role in regulating inflammation. Early assessment of both inflammatory and nutritional markers such as hs-CRP and vitamin D may enhance cardiovascular risk prediction in dyslipidemic patients. Larger studies are needed to confirm these results and assess the benefits of vitamin D supplementation.

KEYWORDS: Dyslipidemia; hs-CRP; Vitamin D; Cardiovascular risk; Inflammation.

INTRODUCTION

Dyslipidemia is a well-established risk factor for cardiovascular diseases and is frequently associated with metabolic disorders such as diabetes mellitus, obesity, and hypertension [1,2]. It plays a central role in the pathogenesis of atherosclerosis and its associated complications [3,4].

Recent evidence has highlighted a potential link between vitamin D deficiency, adverse lipid profiles, and increased cardiovascular risk [5,6]. Dyslipidemia is also commonly accompanied by elevated levels of inflammatory markers, particularly high-sensitivity C-reactive protein (hs-CRP), which serves as a validated biomarker of systemic inflammation and a predictor of cardiovascular events [7,8].

Vitamin D deficiency is now recognized as a global health issue affecting individuals across all age groups [9,10]. Several studies have suggested that low vitamin D levels may contribute to chronic low-grade inflammation and the development of atherosclerosis [11–13]. A large-scale survey confirmed the widespread prevalence of hypovitaminosis D [14]. Furthermore, vitamin D plays a role in modulating immune and inflammatory pathways [15,16,17]. emphasized the role of Vitamin D in immune cell regulation, while Amer and Qayyum [18] and Devaraj et al. [19] reported an inverse association between vitamin D levels and hs-CRP concentrations in large population-based studies.

Given that atherosclerosis involves both lipid dysregulation and chronic inflammation, and that hs-CRP serves as a reliable inflammatory marker, exploring the relationship between vitamin D and hs-CRP in dyslipidemic patients may provide valuable clinical insights [20].

Therefore, the present study was undertaken:

1. To estimate and compare serum concentrations of the inflammatory marker hs-CRP and vitamin D in dyslipidemic patients and healthy controls.

2. To evaluate the correlation between hs-CRP and vitamin D levels in patients with dyslipidemia.

MATERIALS AND METHODS

Ethical Approval: Institutional Ethics Committee (IEC) approval was obtained prior to the initiation of the study. All procedures adhered to the ethical standards of the Declaration of Helsinki.

Study Design: A cross-sectional case-control study was conducted involving 80 subjects:

- **Test group (n = 40):** Patients diagnosed with dyslipidemia as per NCEP-ATP III criteria
- **Control group (n = 40):** Age- and sex-matched healthy individuals

Inclusion Criteria:

- Age between 20–60 years
- Diagnosed dyslipidemia (test group)
- Apparently healthy individuals (control group), matched for age and sex

Exclusion Criteria:

- History of cardiovascular disease, bone disorders, or malabsorption syndromes
- Hepatic or renal dysfunction
- Age < 20 years

Sample Collection and Biochemical Analysis: Five milliliters of venous blood was collected under aseptic precautions. Serum was separated and analyzed for the following parameters:

Table 1: Methods

Parameter	Method	Equipment/Platform
Triglycerides	Enzymatic Method	Dimension-RXL (Siemens)
Total Cholesterol	Cholesterol Oxidase Method	Dimension-RXL
HDL-C	Automated HDL Assay (AHDL)	Dimension-RXL
LDL-C	Automated LDL Assay (ALDL)	Dimension-RXL
hs-CRP	Particle-Enhanced Immunonephelometry	N ProSpec® System [7]
Vitamin D (25(OH)D)	Chemiluminescent Microparticle Immunoassay (CMIA)	Architect Analyzer (Abbott) [9]

Internal Quality Control:

All assays were performed with two-level quality control sera (normal and pathological ranges) supplied by Bio-Rad Laboratories. Quality control was conducted daily to ensure accuracy and reliability.

Statistical Analysis

Data were analyzed using Microsoft Excel and IBM SPSS Statistics for Windows, Version 25.0 (Armonk, NY: IBM Corp.). Continuous variables such as age, lipid parameters (HDL, LDL, total cholesterol, triglycerides), serum Vitamin D, and hs-CRP were expressed as mean ± standard deviation (SD).

1. Descriptive Statistics

Descriptive analyses were used to summarize the central tendency and dispersion of all continuous variables in both dyslipidemia cases and healthy controls (n = 40 per group).

2. Inferential Statistics – Independent Samples t-Test

An independent samples **t-test** was applied to compare mean values of biochemical parameters between the two groups.

- A p-value < 0.05 was considered statistically significant.
- A p-value < 0.001 was considered highly significant.
- The null hypothesis (H_0) assumed no significant difference between the groups.

3. Correlation Analysis – Pearson's Correlation Coefficient (r)

Pearson's correlation coefficient was used to assess the relationship between Vitamin D and hs-CRP levels within each group. Correlation strength and direction were interpreted using standard guidelines, and significance was determined at $p< 0.05$.

Table 2: Significance Symbols

Symbol	Meaning
NS	Not Significant (p> 0.05)
S	Significant (p< 0.05)
HS	Highly Significant (p< 0.001)

RESULTS

Demographic Analysis

The study comprised a total of 80 subjects, divided equally into a Control group (n = 40) and a Test group (n = 40). In the control group, 19 subjects (47.5%) were female and 21 (52.5%) were male. The test group included 23 females (57.5%) and 17 males (42.5%). This suggests a reasonably balanced gender distribution across both groups, with no statistically significant difference (Chi-square test, p = 0.5018).

The mean age of the control group was 37.88 ± 12.29 years (range: 20–59), while the test group had a mean age of 41.25 ± 12.04 years (range: 21–58). The age distribution was also not significantly different between groups (unpaired t-test, p = 0.2185).

Table 3: Demographic Characteristics of Study Participants

Group	Sample Size (n)	Female (n, %)	Male (n, %)	Mean Age ± SD (years)	Age Range (years)
Control	40	19 (47.5%)	21 (52.5%)	37.88 ± 12.29	20–59
Test	40	23 (57.5%)	17 (42.5%)	41.25 ± 12.04	21–58

Note: Both groups were age- and sex-matched. No statistically significant differences were observed between groups (see Table 4).

(See Figure 1 for visual representation.)

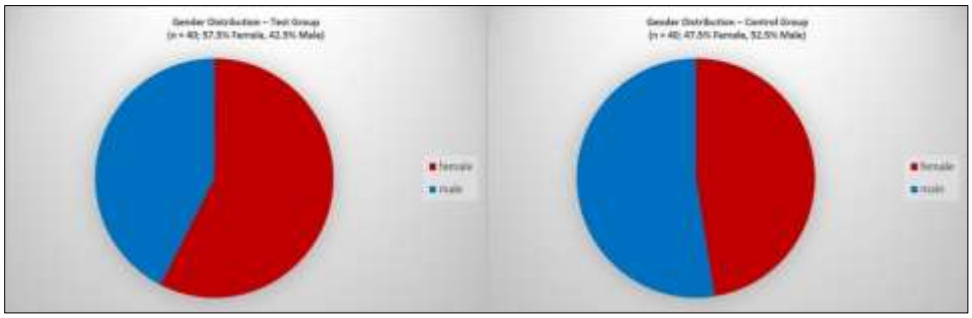


Figure 1: Gender distribution of study participants in the test and control groups.
(Pie chart showing percentage distribution of male and female subjects in both groups)

Pie charts show the percentage of male and female participants in each group (n = 40 per group). The test group included 57.5% females and 42.5% males, while the control group comprised 47.5% females and 52.5% males. The difference in gender distribution between the groups was not statistically significant (p = 0.5018; Chi-square test).
(See Figure 2 for visual representation.)

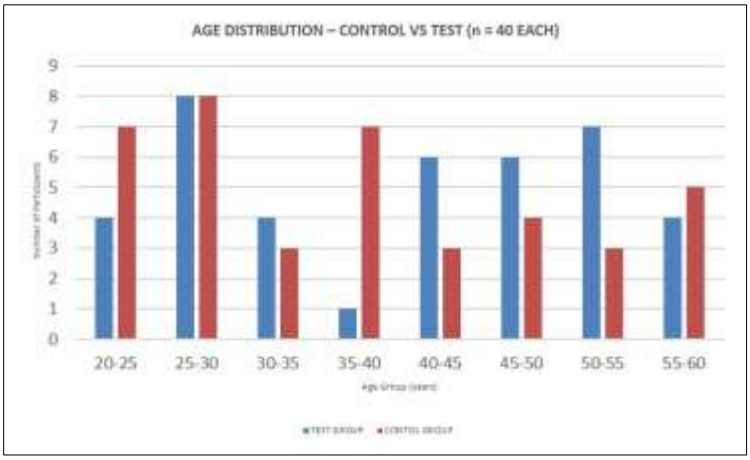


Figure 2: Age Distribution of Study Participants

Figure 2. Grouped bar chart showing age distribution across control and test groups (n = 40 each). Although minor differences were observed in specific age brackets (e.g., 35–40 and 50–55 years), the overall mean age difference between the two groups was not statistically significant (Unpaired t-test, p = 0.2185).

Table 4: Statistical Test Results for Demographic Comparability

Test	Test Statistic	p-value	Interpretation	Significance
Unpaired t-test (Age)	–1.241	0.2185	No significant difference in age	NS (p > 0.05)
Chi-square test (Gender)	0.451	0.5018	No significant difference in gender	NS (p > 0.05)

NS: Not Significant (p > 0.05). This confirms that age and gender distributions do not differ significantly between groups.

Significance of Demographic Analysis

The demographic analysis is critical to the methodological soundness and internal validity of the study. Its importance is highlighted below:

1. **Baseline Comparability:** Both age and gender distributions were statistically comparable across groups. This ensures that observed biochemical differences are not confounded by demographic variation.
2. **Minimization of Selection Bias:** Age- and sex-matching, validated by statistical tests, reduces selection bias and enhances internal validity.
3. **Strengthens Biochemical Interpretation:** With demographic parity confirmed, the observed variations in Vitamin D and hs-CRP are more confidently attributed to dyslipidemic status rather than confounding.
4. **Supports Valid Correlation Analysis:** Since age and sex can independently affect both Vitamin D and hs-CRP levels, confirming comparability ensures the inverse correlation observed in the dyslipidemia group (r = –0.36, p = 0.021) is robust.

Table 5: Comparison of Biochemical Parameters Between Dyslipidemia Cases and Controls (n = 40 each)

Parameter	Group	Mean ± SD	p-value	Significance
HDL (mg/dL)	Test	38.83 ± 19.15	0.008	HS
	Control	47.28 ± 4.93		
LDL (mg/dL)	Test	119.05 ± 52.35	0.001	HS
	Control	70.20 ± 22.67		
Total Cholesterol (mg/dL)	Test	207.63 ± 52.35	0.0001	HS
	Control	134.75 ± 22.67		
Triglycerides (mg/dL)	Test	281.88 ± 134.89	0.001	HS
	Control	86.35 ± 28.12		
Vitamin D (ng/mL)	Test	14.57 ± 6.48	0.001	HS
	Control	21.49 ± 11.10		
hs-CRP (mg/L)	Test	10.42 ± 10.47	0.001	HS
	Control	2.29 ± 1.72		

SD – Standard Deviation; **HDL** – High-Density Lipoprotein; **LDL** – Low-Density Lipoprotein; **hs-CRP** – High-sensitivity C-Reactive Protein; **HS** – Highly Significant (p< 0.001). Statistical analysis was performed using the independent samples t-test.

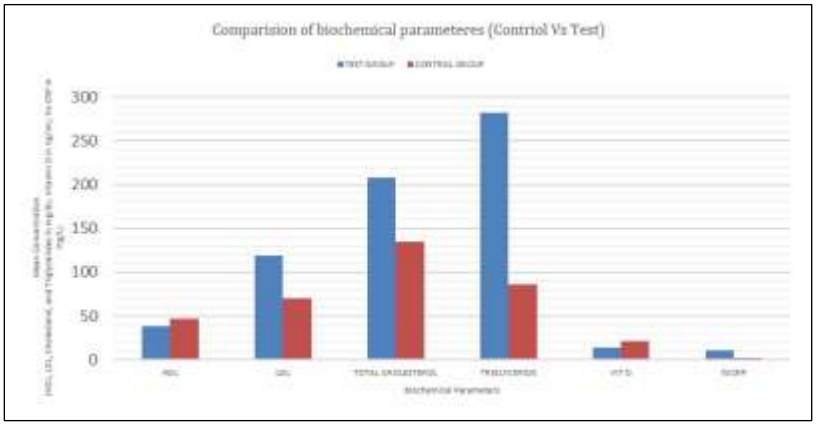


Figure 3: Comparision of biochemical parameters

Figure 3. Bar chart showing mean values of biochemical parameters (HDL, LDL, Total Cholesterol, Triglycerides, Vitamin D, and hs-CRP) in test and control groups (n = 40 each). Dyslipidemic individuals (test group) exhibited

higher levels of LDL, total cholesterol, triglycerides, and hs-CRP, and lower Vitamin D and HDL levels compared to controls.

Interpretation of Biochemical Parameter Comparison

- **HDL (High-Density Lipoprotein):**
  ↓ Significantly lower in the dyslipidemia group (p = 0.008, **S**)
  → Suggests reduced cardioprotective potential.
- **LDL (Low-Density Lipoprotein):**
  ↑ Markedly elevated in dyslipidemic subjects (p = 0.001, **HS**)
  → Confirms enhanced atherogenic profile.
- **Total Cholesterol:**
  ↑ Significantly higher in cases (p = 0.0001, **HS**)
  → Indicates overall lipid imbalance.
- **Triglycerides (TG):**
  ↑ Extremely elevated in test group (p = 0.001, **HS**)
  → Strongly associated with metabolic syndrome.
- **Vitamin D:**
  ↓ Notable deficiency in dyslipidemic patients (p = 0.001, **HS**)
  → Highlights its possible anti-inflammatory and lipid-modulating role.
- **hs-CRP (High-Sensitivity C-Reactive Protein):**
  ↑ Substantially higher in the dyslipidemia group (p = 0.001, **HS**)
  → Indicates systemic low-grade inflammation.
- Comparison of Covariance, Correlation, and Significance of Vitamin D and hs-CRP (Test vs Control)

Table 6: Comparison of Covariance, Correlation, and Significance of Vitamin D and hs-CRP (Test vs Control

Measure	Test Group	Control Group
Covariance (cov)	–24.0331	–1.8900
Mean Vitamin D	6.4755	11.0994
Mean hs-CRP	10.4699	1.7226
Correlation (r)	–0.3545	–0.10
p-value	**0.024** (significant)	**0.539** (ns)

Interpretation Summary:

1. **Covariance**
   - In the test group (dyslipidemia patients), the covariance between Vitamin D and hs-CRP is –24.03, indicating that as Vitamin D levels decrease, hs-CRP levels tend to increase substantially.
   - In the control group, the covariance is much smaller (–1.89), showing a weak inverse relationship.
2. **Mean Values**
   - Vitamin D is markedly lower in the test group (6.48 ng/mL) compared to controls (11.10 ng/mL), suggesting Vitamin D deficiency is more pronounced in dyslipidemia patients.
   - hs-CRP, an inflammatory marker, is substantially higher in the test group (10.47 mg/L) than in controls (1.72 mg/L), indicating greater systemic inflammation in dyslipidemia patients.
3. **Correlation (r)**
   - Test group: A moderate negative correlation (r = –0.3545) means lower Vitamin D levels are moderately associated with higher hs-CRP levels.
   - Control group: Very weak negative correlation (r = –0.10), showing little association between Vitamin D and hs-CRP.
4. **Statistical Significance (p-value)**
   - In the test group, p = 0.024 (< 0.05), so the negative correlation between Vitamin D and hs-CRP is statistically significant.
   - In the control group, p = 0.539 (> 0.05), meaning the relationship is not statistically significant.

Overall Conclusion

In dyslipidemia patients, lower Vitamin D levels are significantly associated with higher systemic inflammation (as measured by hs-CRP). This relationship is absent in healthy controls, suggesting Vitamin D deficiency may contribute to inflammatory processes in dyslipidemia.

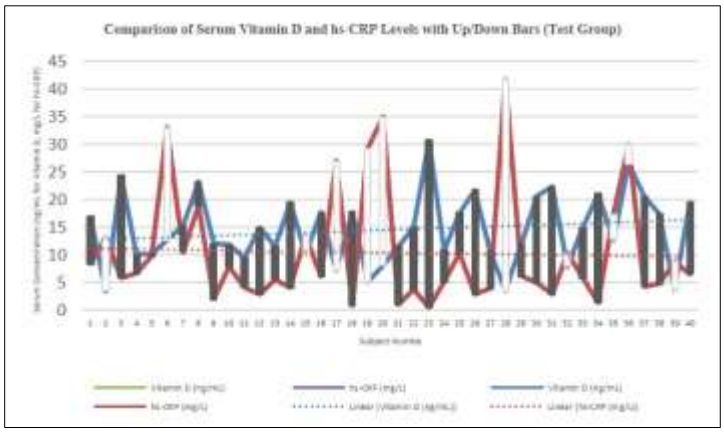
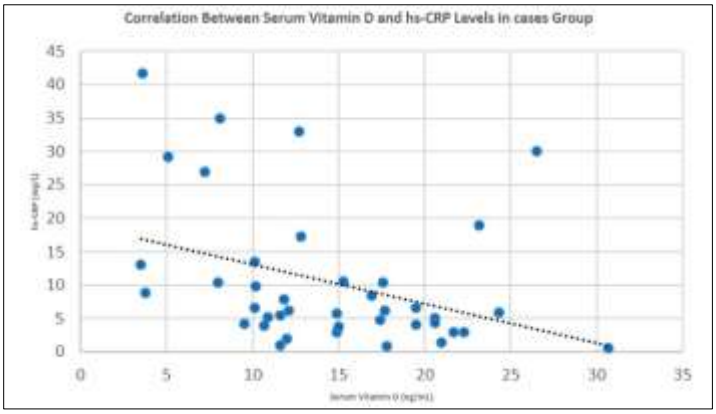


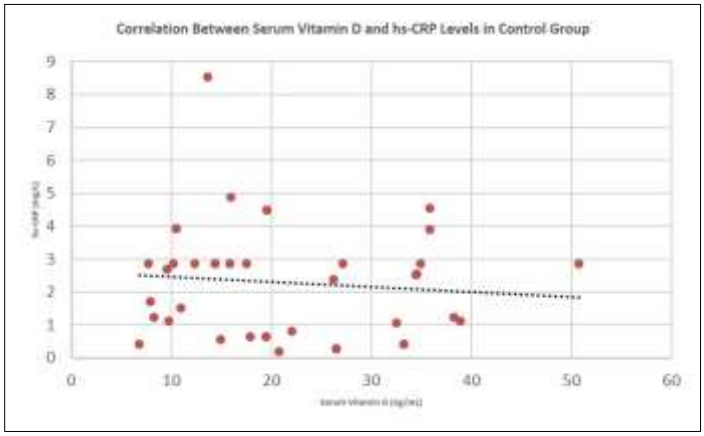
Figure 4A: Comparison of Serum Vitamin D and hs-CRP Levels with Up/Down Bars (Test Group)

(4A) Line chart with up/down bars showing individual-level comparison of Vitamin D and hs-CRP concentrations across test group participants. The up/down bars highlight the difference between the two markers for each subject, indicating a general inverse pattern where higher CRP tends to coincide with lower Vitamin D.



4B: Scatter plot -Correlation Between Vitamin D and hs-CRP Level (Test group)

(B)Scatter plot representing the correlation between serum Vitamin D and hs-CRP levels in the test group. A moderate negative correlation (r = –0.3545) is observed, suggesting that individuals with lower Vitamin D levels tend to exhibit higher levels of systemic inflammation, as indicated by hs-CRP. This supports a potential inverse relationship between Vitamin D status and inflammatory response in dyslipidemic individuals.



4C: Scatter plot -Correlation Between Vitamin D and hs-CRP Level (Control group)

(4C) Scatter plot for the control group, showing a weak negative correlation (r = –0.10) between Vitamin D and hs-CRP levels. The flatter trendline and more dispersed data points indicate minimal or no association in apparently healthy individuals, in contrast to the test group.

Correlation Analysis Summary
Dyslipidemia Group:

- Pearson's r = –0.3545, p = 0.024
- Moderate inverse and statistically significant correlation
- → Suggests that lower Vitamin D levels are associated with higher hs-CRP concentrations, indicating a potential anti-inflammatory role of Vitamin D in dyslipidemia.

Control Group:

- **Pearson's r = –0.10**, **p = 0.539**
- Weak and **not statistically significant** correlation
- → Implies that the inverse relationship between Vitamin D and hs-CRP is disease-specific and not observed in healthy individual

Collective Summary
This study demonstrated the following findings in dyslipidemia patients:

- **Dysregulated lipid profile:** ↑ LDL, ↑ Triglycerides, ↑ Total Cholesterol, ↓ HDL
- **Deficient Vitamin D status:** → Suggestive of metabolic and immunoregulatory imbalance
- **Elevated systemic inflammation:** ↑ hs-CRP levels
- **Significant inverse correlation between Vitamin D and hs-CRP:** → Indicates that Vitamin D deficiency may exacerbate inflammation, reinforcing its clinical value as a biomarker and possible therapeutic target in managing dyslipidemia-associated cardiovascular risk.

DISCUSSION
findings underscore the strong relationship between dyslipidemia and the development of atherosclerosis and cardiovascular complications [2]. In our cohort, patients with dyslipidemia exhibited elevated levels of LDL, total cholesterol, and triglycerides, along with a reduction in HDL levels. These findings are consistent with the well-established lipid abnormalities that contribute to cardiovascular risk [3,4].

The inflammatory nature of atherosclerosis has brought increasing attention to biomarkers such as high-sensitivity C-reactive protein (hs-CRP). According to CDC/AHA guidelines, hs-CRP is a validated inflammatory biomarker for cardiovascular risk stratification [7,8]. The increased hs-CRP concentrations observed in the dyslipidemia group further highlight the contribution of persistent inflammation in such individuals.

Studies by Ouchi et al. [11], Katsiki et al. [12], and Saltiel and Olefsky [13] corroborate our findings, demonstrating increased inflammatory activity in individuals with metabolic syndrome and dyslipidemia.

Vitamin D deficiency was found to be notably more common among dyslipidemia patients in our study. Similar findings were reported by Holick [9,14], who emphasized the global prevalence of hypovitaminosis D. Mangin et al. [15] and Liu et al. [16] also suggested that Vitamin D plays a regulatory role in immune function and inflammation.

This is further supported by Calton et al. [17], who emphasized the immunomodulatory potential of Vitamin D in cellular models, and by observational data from Amer and Qayyum [18] and Devaraj et al. [19], showing an inverse association between serum 25(OH)D and hs-CRP in diabetic and non-diabetic populations.

Several studies have reported a favorable influence of Vitamin D on lipid parameters, especially total cholesterol and LDL. Jorde and Grimnes [21] as well as Wang et al. [22] demonstrated modest improvements in lipid profiles following Vitamin D supplementation.

Importantly, we observed a statistically significant inverse relationship between Vitamin D and hs-CRP levels in patients with dyslipidemia. This supports the hypothesis that Vitamin D functions as an anti-inflammatory agent. Our findings align with prior studies by Tao et al. [23] and Luo et al. [24], who demonstrated an inverse association between serum Vitamin D levels and inflammatory markers such as hs-CRP.

Vitamin D is known to exert anti-inflammatory effects by modulating immune responses. As shown by DeLuca and Cantorna [25], it downregulates the expression of pro-inflammatory cytokines such as IL-6 and TNF-α while upregulating anti-inflammatory mediators. This immunoregulatory role suppresses hepatic CRP synthesis, leading to lower circulating hs-CRP levels [26]. Thus, adequate Vitamin D status may help mitigate chronic low-grade inflammation, a key feature of atherosclerosis and metabolic syndrome.

Recent studies support these associations. Charoenngam et al. [27] reported that Vitamin D supplementation reduced hs-CRP levels in healthy adults. Similarly, Kamble et al. [28] demonstrated improvement in inflammatory markers and insulin sensitivity in patients with type 2 diabetes. Lee et al. [29] confirmed that Vitamin D deficiency was associated with elevated CRP levels in Korean adults.

Collectively, these findings strengthen the hypothesis that Vitamin D deficiency contributes to systemic inflammation in dyslipidemia and may represent a modifiable risk factor in cardiovascular disease prevention. The inverse relationship observed between hs-CRP and Vitamin D suggests that optimizing Vitamin D status could have significant anti-inflammatory and cardioprotective benefits in dyslipidemic populations.

CONCLUSION

This study highlights important findings about the inflammation and vitamin D status in people with dyslipidemia. We found that patients with dyslipidemia had higher levels of hs-CRP, indicating more inflammation, and lower levels of vitamin D. This suggests that low vitamin D may play a role in increasing inflammation and cardiovascular risk.

There was a moderate inverse correlation between hs-CRP and vitamin D, which supports the idea that vitamin D may help reduce inflammation. Research shows that vitamin D can lower pro-inflammatory molecules like IL-6 and TNF-α, and reduce CRP production in the liver.

Since vitamin D deficiency is commonly linked to heart and metabolic problems, regularly checking and correcting vitamin D levels in people with dyslipidemia may help reduce inflammation and long-term cardiovascular risk. However, because this was a cross-sectional study, we cannot prove a cause-and-effect relationship.

Larger and longer-term studies are needed to confirm these findings and explore whether vitamin D supplements can help in treating dyslipidemia. Using both traditional lipid markers and newer indicators like hs-CRP and vitamin D may improve risk prediction and allow for more personalized care.

Based on our results and similar global findings, routine screening for vitamin D in dyslipidemic patients may be a simple and cost-effective way to reduce inflammation and prevent heart disease.

DECLARATIONS

Ethical Clearance

The study was approved by the Institutional Ethics Committee of Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune (IEC No.: XYZ/2024/007), in accordance with the Declaration of Helsinki.

Consent for Publication

Not applicable. No individual identifiable data (such as images, videos, or clinical photographs) are included in this manuscript.

Competing Interests: The author declares no competing interests.

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Authors' Contributions: Dr. Sandesh Thorat is the sole author of this study. He conceptualized and designed the research, performed the data analysis, interpreted the results, and wrote the manuscript.

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The authors further declare that artificial intelligence tools (such as ChatGPT, OpenAI) were used solely for language editing, grammar correction, and formatting purposes. All scientific content, interpretations, and conclusions are the original work of the authors, who take full responsibility for the integrity and accuracy of the final manuscript. Special thanks to Miss Mansi Tomar, MBBS student, for her valuable assistance in data collection during the study.

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