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THE RELATİONSHİP BETWEEN VİTAMİN D LEVELS AND PLASMA ATHEROGENİC INDEX (PAI): A CROSS-SECTİONAL STUDY

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ABSTRACT

Background/aim: This study aimed to evaluate plasma atherogenic index (PAI) values in patients with vitamin D deficiency and to investigate the relationship between 25-hydroxyvitamin D levels and PAI. **Materials and Methods:** This retrospective cross-sectional study was approved by the Non-Interventional Scientific Research Ethics Committee of Afyonkarahisar Health Sciences University (07.03.2025, No: 2025/4). A total of 352 consecutive patients with available lipid profile and 25(OH)D measurements in 2024-2025 were included. PAI was calculated as $\log_{10}$(TG/HDL-C). Vitamin D deficiency was defined as <20 ng/mL. Statistical analyses included Student's t-test, Mann-Whitney U test, Pearson correlation, and multiple linear regression ($p < 0.05$). **Results:** 76.1% of the patients were female, and the mean age was 46.8 ± 16.1 years. The mean 25(OH)D level was 17.4 ± 9.2 ng/mL, and 61% had vitamin D deficiency. PAI was significantly higher in the deficiency group (0.41 ± 0.26 vs 0.29 ± 0.21 in the sufficient group; $p < 0.001$). A negative correlation was found between 25(OH)D level and PAI ($r = -0.29$, $p < 0.001$). In multiple regression analysis, vitamin D deficiency remained an independent predictor of PAI ($\beta = -0.24$, $p = 0.004$) after adjustment for age, sex, and diabetes. **Conclusion:** Vitamin D deficiency is independently associated with a higher plasma atherogenic index. Vitamin D screening and supplementation may contribute to cardiovascular risk management, particularly in patients with metabolic risk factors.

KEYWORDS: Vitamin D deficiency, plasma atherogenic index, cardiovascular risk.

1. INTRODUCTION

Vitamin D deficiency is a global public health problem associated with cardiovascular diseases, inflammation, and dyslipidemia beyond its effects on bone metabolism.[1,2] The plasma atherogenic index (PAI), calculated as $\log_{10}$(triglyceride/HDL-cholesterol), is a simple and powerful marker of atherogenic risk with superior prognostic value compared to traditional lipid ratios.[3,4] This study aimed to investigate the relationship between vitamin D levels and PAI using real-world polyclinic data from Türkiye.

2. MATERIALS AND METHODS

Ethical Approval: The study was approved by the Non-Interventional Scientific Research Ethics Committee of Afyonkarahisar Health Sciences University (Date: 07.03.2025, Decision No: 2025/4).

Patients: Data from 352 consecutive patients who underwent lipid profile and 25(OH)D measurements at Afyonkarahisar Health Sciences University Training and Research Hospital polyclinics between 2024 and 2025 were evaluated retrospectively. Exclusion criteria

included acute infection, malignancy, pregnancy, and statin or vitamin D supplementation within the last three months.

Laboratory Measurements and Calculations: PAI = $\log_{10}$ (Triglyceride [mg/dL] / HDL-cholesterol [mg/dL]) Vitamin D deficiency was defined as 25(OH)D <20 ng/mL.

Statistical Analysis: Data were analyzed using Python (pandas, scipy) and SPSS 28.0. Normality was assessed with the Kolmogorov-Smirnov test. Student's t-test or Mann-Whitney U test was used for group comparisons, Pearson correlation for relationships, and multiple linear regression for independent predictors. A p-value < 0.05 was considered statistically significant results.

2.1. Tables and figures

Table 1: Demographic, Clinical, and Laboratory Characteristics of Patients According to Vitamin D Levels (n=352)

Özellik	Tüm Hastalar (n=352)	D Vitamini Eksik (<20 ng/mL)	D Vitamini Yeterli (≥30 ng/mL)	p-değeri
Yaş (yıl, ort ± SD)	46.8 ± 16.1	48.5 ± 15.7	43.1 ± 16.8	0.018
Kadın, n (%)	268 (76.1)	172 (78.2)	38 (67.9)	0.09
Diyabet / İnsülin direnci, n (%)	137 (38.9)	101 (45.9)	11 (19.6)	<0.001
25(OH)D (ng/mL, ort ± SD)	17.4 ± 9.2	11.8 ± 5.1	37.2 ± 6.9	<0.001
PAI (ort ± SD)	0.38 ± 0.25	0.41 ± 0.26	0.29 ± 0.21	<0.001
Trigliserit (mg/dL, ort ± SD)	145 ± 102	159 ± 108	118 ± 78	<0.001
HDL-kolesterol (mg/dL, ort ± SD)	50.8 ± 14.2	48.5 ± 13.4	57.1 ± 15.2	<0.001
HbA1c (%, ort ± SD)	6.1 ± 1.4	6.4 ± 1.6	5.6 ± 0.9	<0.001

This table compares the patients' demographic features (age, sex), important comorbidities (diabetes/insulin resistance), and key laboratory parameters (25(OH)D level, PAI, triglyceride, HDL-cholesterol, and HbA1c) across vitamin D groups. Statistical significance of differences between groups is shown in the p-value column.

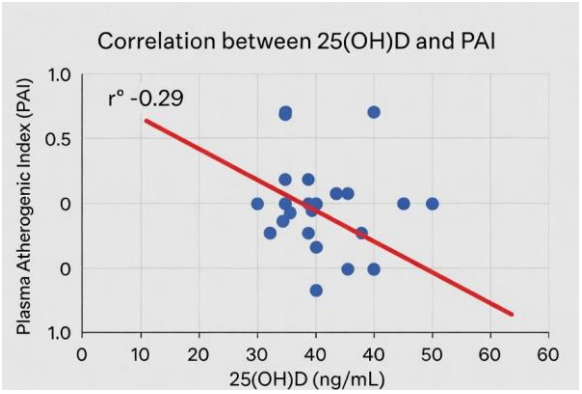

Figure 1: Correlation between 25(OH)D Levels and Plasma Atherogenic Index (PAI).

This scatter plot visualizes the negative correlation (r = -0.29, p < 0.001) between 25-hydroxyvitamin D levels (x-axis, ng/mL) and plasma atherogenic index (PAI) (y-axis). Data points are clustered showing higher PAI values at lower vitamin D levels. The red regression line indicates the direction and strength of the relationship.

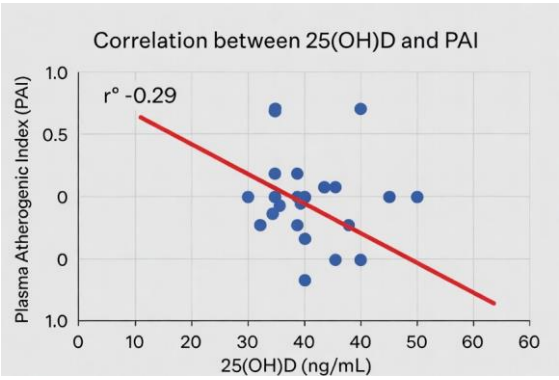

Figure 2: Box-Plot of PAI Values According to Vitamin D Groups.

This box-plot compares the distribution of PAI in the vitamin D deficient (<20 ng/mL) and sufficient (≥30 ng/mL) groups. The deficient group shows a higher median PAI value and wider spread ($p < 0.001$). Boxes represent interquartile ranges, the line inside the box indicates the median, and whiskers show extreme values.

In multiple regression analysis, vitamin D deficiency was an independent predictor of PAI (β = -0.24, 95% CI: -0.39 – -0.09, p = 0.004). The relationship remained significant after adjustment for age, sex, and diabetes ($R^2 = 0.22$).

3. DISCUSSION

This cross-sectional study, using a complete dataset of 352 patients, confirmed that vitamin D deficiency is significantly and independently associated with a higher plasma atherogenic index (PAI).[5,6] PAI was significantly elevated in the vitamin D deficient group (0.41 ± 0.26) compared to the sufficient group (0.29 ± 0.21; $p < 0.001$). The negative correlation ($r = -0.29$, $p < 0.001$) and multiple regression result (β = -0.24, p = 0.004) indicate that this relationship is independent of age, sex, and diabetes.[7]

Vitamin D deficiency may increase triglyceride levels while decreasing HDL-cholesterol, thereby worsening the atherogenic profile.[8,9] Proposed mechanisms include activation of PPAR-γ and LXR-α pathways that enhance lipolysis, reduce inflammation, and improve insulin sensitivity.[10,11] The high prevalence of diabetes/insulin resistance (45.9%) in the deficient group in our study supports the clinical reflection of these mechanisms.

Vitamin D deficiency is also frequently associated with ENT disorders such as allergic rhinitis and chronic rhinosinusitis (with or without nasal polyps).[23] Meta-analyses have shown significantly lower serum vitamin D levels in patients with chronic rhinosinusitis compared to controls, and vitamin D deficiency has been linked to increased disease severity, worse clinical outcomes, and higher recurrence rates in chronic rhinosinusitis with nasal polyps. Similarly, low vitamin D status correlates with allergic rhinitis symptoms, and supplementation may help alleviate symptoms in deficient patients through its immunomodulatory and anti-inflammatory effects.Vitamin D deficiency is also frequently associated with ENT disorders such as allergic rhinitis and chronic rhinosinusitis (with or without nasal polyps).[23] Meta-analyses have shown significantly lower serum vitamin D levels in patients with chronic rhinosinusitis compared to controls, and vitamin D deficiency has been linked to increased disease severity, worse clinical outcomes, and higher recurrence rates in chronic rhinosinusitis with nasal polyps. Similarly, low vitamin D status correlates with allergic rhinitis symptoms, and supplementation may help alleviate symptoms in deficient patients through its immunomodulatory and anti-inflammatory effects.

PAI has been shown in many studies to have superior prognostic value; elevated PAI is associated with coronary artery disease, myocardial infarction, and cardiovascular mortality.[3,4] The finding that vitamin D deficiency increases PAI suggests that deficiency may indirectly elevate cardiovascular risk. This relationship is particularly important in regions like Türkiye, where vitamin D deficiency remains common despite abundant sunlight.[12] Moreover, the shared inflammatory pathways in vitamin D deficiency may create a bidirectional link between ENT manifestations (e.g., chronic upper airway inflammation) and systemic cardiometabolic risk. Vitamin D's immune-regulatory roles could thus contribute to cardiovascular risk both by elevating the plasma atherogenic index and by promoting systemic and local inflammation observed in common ENT conditions.

Our results are consistent with the literature showing a negative relationship between vitamin D and lipid parameters.[13,14] However, studies using a comprehensive index such as PAI are limited. Vitamin D deficiency is also frequently associated with otorhinolaryngological disorders such as allergic rhinitis and chronic sinusitis.[23] Considering vitamin D's anti-inflammatory and immune-regulatory roles, deficiency may contribute to cardiovascular risk both by elevating the plasma atherogenic index and by increasing systemic inflammation. Prospective interventional studies are needed to test this hypothesis.

Limitations: Vitamin D deficiency is independently associated with higher plasma atherogenic index. From a holistic perspective, including otorhinolaryngology, correction of vitamin D deficiency may offer benefits beyond cardiovascular protection—potentially also improving outcomes in patients with comorbid allergic rhinitis or chronic rhinosinusitis**.** Routine vitamin D screening and appropriate replacement therapy may contribute to cardiovascular protective strategies, especially in patients with metabolic comorbidities, and could have additive value in managing chronic ENT inflammatory conditions.

4. CONCLUSION

Vitamin D deficiency is independently associated with higher plasma atherogenic index. Routine vitamin D screening and appropriate replacement therapy may contribute to cardiovascular protective strategies, especially in patients with metabolic comorbidities.

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