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ORIGINAL ARTICLE

Vitamin D levels and their relationship with lipid metabolism and inflammatory markers in healthy adults

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Abstract

Objective: This study investigates the associations between serum vitamin D levels and biochemical, hematological, and inflammatory markers in a healthy adult population, focusing on their implications for lipid metabolism and systemic inflammation.

Methods: A retrospective analysis was conducted on the medical records of 267 individuals aged 18–65 years, who presented to the internal medicine outpatient clinic with complaints of fatigue. Inclusion criteria required participants to be free of any chronic diseases, acute medical conditions. Serum vitamin D levels were categorized into three groups: deficient-insufficient (≤19 ng/mL), sufficient (20–29 ng/mL), and normal (≥30 ng/mL). Biochemical parameters, hematological markers, and derived ratios (e.g., triglyceride/glucose ratio, monocyte/HDL-C ratio) were analyzed. Spearman correlation, Kruskal-Wallis H test, and Mann-Whitney U test were used for statistical analysis.

Results: Vitamin D levels showed significant positive correlations with HDL-C ($\rho = 0.169$, $p = 0.0055$) and LDL-C ($\rho = 0.198$, $p = 0.0011$), and a negative correlation with neutrophil counts ($\rho = -0.133$, $p = 0.030$). Among derived ratios, the triglyceride/monocyte ratio exhibited a significant positive correlation ($\rho = 0.160$, $p = 0.0088$), while the monocyte/HDL-C ratio showed a significant negative correlation ($\rho = -0.203$, $p = 0.00083$). Group comparisons revealed significantly lower HDL-C levels in the deficient-insufficient group compared to the sufficient and normal groups ($p = 0.0022$). No significant differences were found for other lipid or inflammatory markers.

Conclusion: This study highlights the multifaceted roles of vitamin D in lipid metabolism and systemic inflammation. The positive association with HDL-C underscores its potential cardioprotective effects, while the negative correlation with neutrophil counts suggests its role in modulating inflammation. These findings provide valuable insights into vitamin D's broader physiological effects in healthy individuals, warranting further large-scale studies.

Keywords: Vitamin D; lipid metabolism; inflammation; biomarkers

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Introduction

Vitamin D is a fat-soluble steroid derivative that influences the intestines, bones, kidneys, and parathyroid glands. It plays a fundamental role in bone mineralization and the regulation of calcium and phosphorus levels [1,2]. Recent studies have highlighted its broader impact, extending beyond these classical roles to include significant effects on metabolic and immunological processes.

Epidemiological studies estimate that approximately one billion people worldwide suffer from vitamin D deficiency. The prevalence varies by geographic location, duration of sun exposure, use of sunscreen, and dietary habits [3]. The deficiency is particularly pronounced in adults residing in the Middle East and Asia [4].

The best indicator of vitamin D status is its circulating metabolite, 25(OH)D, which has a half-life of 10–19 days. This metabolite reflects the amount of vitamin D synthesized in the skin through ultraviolet exposure or obtained from dietary sources. In the liver, vitamin D is converted to 25(OH)D, its primary circulating form, by 25-hydroxylase enzymes. Subsequently, 25(OH)D is metabolized in the kidneys to its active form, 1,25-dihydroxyvitamin D3 (calcitriol), via the enzyme 1-alpha-hydroxylase. This active form is also produced extrarenally in response to cytokines such as tumor necrosis factor-alpha and interferon-gamma, highlighting its role in immune regulation [5,6].

Vitamin D deficiency has been associated with a variety of diseases and metabolic disorders. Studies have shown that vitamin D supplementation can improve markers of metabolic health, including reductions in total cholesterol, low-density lipoprotein (LDL), triglycerides (TG), glycated hemoglobin (HbA1c), and HOMA-IR, an indicator of insulin resistance, particularly in individuals with type 2 diabetes mellitus [7,8]. However, the precise mechanisms underlying these effects remain unclear.

Research has demonstrated that vitamin D receptors and enzymes involved in vitamin D metabolism are expressed in various cells, including insulin-sensitive pancreatic beta cells and adipocytes. Adipose tissue, which serves as a storage site for vitamin D, also secretes adipokines and cytokines that actively contribute to systemic inflammation [9,10]. These findings suggest a complex interplay between vitamin D, inflammation, and metabolic health.

In addition to its well-established roles in calcium and bone metabolism, vitamin D exerts non-classical effects on other physiological systems. It modulates immune responses, influences lipid and glucose metabolism, and reduces systemic inflammation. These effects are particularly relevant to conditions such as diabetes, atherosclerosis, and autoimmune diseases [11-14].

Recent studies have also identified novel biomarkers, such as the triglyceride/glucose ratio, triglyceride/HDL-C ratio, and monocyte/HDL-C ratio, which provide insights into the relationships between lipid metabolism, glucose regulation, and immune responses. These markers may offer new perspectives on the broader physiological roles of vitamin D, particularly in metabolic and cardiovascular health [15,16].

Given these extensive roles, this study aims to investigate the relationships between vitamin D levels, glucose metabolism, lipid profiles, and inflammatory markers in a healthy population. By focusing on key biomarkers, we seek to provide new insights into the systemic effects of vitamin D, contributing to a more comprehensive understanding of its role in maintaining metabolic balance.

Methods

Approval for this study was obtained from the ethics committee of our hospital. We retrospectively reviewed the medical records of 2790 individuals who presented with complaints of fatigue to the internal medicine outpatient clinic of our hospital between October 1, 2022, and December 31, 2022. After applying the inclusion and exclusion criteria, a total of 267 participants were included in the final analysis. Inclusion criteria were as follows: patients aged 18–65 years with no active disease. Exclusion criteria included the presence of comorbid diseases, regular medication use, glucose levels >125 mg/dL, and abnormal TSH levels (>4.5 or <0.5).

Laboratory parameters, including biochemical and hematological markers, were obtained from patient records as part of the retrospective analysis. Biochemical parameters included glucose (mg/dL), triglycerides (mg/dL), low-density lipoprotein cholesterol (LDL-C, mg/dL), and high-density lipoprotein cholesterol (HDL-C, mg/dL). Hematological parameters included neutrophil count ($\times 10^3/\mu L$), lymphocyte count ($\times 10^3/\mu L$), monocyte count ($\times 10^3/\mu L$), and platelet count ($\times 10^3/\mu L$). These parameters were measured using standard

automated biochemical and hematological analyzers at the hospital's central laboratory.

In addition to these primary laboratory parameters, derived ratios such as the triglyceride/glucose ratio (triglyceride [mg/dL] ÷ glucose [mg/dL]), triglyceride/HDL-C ratio (triglyceride [mg/dL] ÷ HDL-C [mg/dL]), triglyceride/monocyte ratio (triglyceride [mg/dL] ÷ monocyte count [$\times 10^3/\mu L$]), monocyte/HDL-C ratio (monocyte count [$\times 10^3/\mu L$] ÷ HDL-C [mg/dL]), neutrophil/lymphocyte ratio (neutrophil count [$\times 10^3/\mu L$] ÷ lymphocyte count [$\times 10^3/\mu L$]), and platelet/lymphocyte ratio (platelet count [$\times 10^3/\mu L$] ÷ lymphocyte count [$\times 10^3/\mu L$]) were calculated to further assess metabolic and inflammatory markers.

Serum vitamin D levels were measured using the chemiluminescence immunoassay (CLIA) method with an automated analyzer (Roche Cobas e601). This method is widely used for its high sensitivity and specificity in the quantification of 25-hydroxyvitamin D [25(OH)D] levels. Vitamin D levels were extracted from patient records. Participants were categorized into three groups based on their serum vitamin D levels:

- Group 1: Deficient (<10 ng/mL)
- Group 2: Insufficient (10–19.9 ng/mL)
- Group3:Sufficient(≥20ng/mL)

Statistical Analysis

All statistical analyses were conducted using the Statistical Package for Social Sciences (SPSS), Version 25 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize the demographic and clinical characteristics of the study participants. These statistics included frequency distributions, percentages, median values, interquartile ranges (IQR), means, and standard deviations (SD).

The normality of the data distribution was assessed using the Shapiro-Wilk test. Since the data did not meet the criteria for normal distribution, non-parametric statistical tests were employed.

The correlations between vitamin D levels and biochemical parameters (LDL-C, HDL-C, triglycerides, glucose, neutrophil, lymphocyte, monocyte, and platelet levels) as well as derived ratios (triglyceride/glucose ratio, triglyceride/HDL-C ratio, triglyceride/monocyte ratio, monocyte/HDL-C ratio, neutrophil/lymphocyte ratio, and platelet/lymphocyte ratio) were examined using the Spearman correlation analysis. Correlation coefficients (ρ) and p-values were reported.

Patients were categorized into three groups based on their serum vitamin D levels: Group 1 (≤19 ng/mL), Group 2 (20–29 ng/mL), and Group 3 (≥30 ng/mL). Differences in biochemical parameters and derived ratios among the groups were analyzed using the Kruskal-Wallis H test for continuous variables. Pairwise comparisons among groups for statistically significant parameters were conducted using the Mann-Whitney U test.

To account for potential confounding variables, a subgroup analysis was performed by excluding patients with folate levels ≤3 ng/mL, vitamin B12 levels ≤250 pg/mL, and iron levels ≤60 µg/dL. Statistical analyses for this subgroup followed the same methodology as described above.

For all tests, a p-value of <0.05 was considered statistically significant.

Results

A total of 267 individuals, including 191 women (71.5%) and 76 men (28.5%), were included in the study based on the inclusion criteria. The demographic and clinical characteristics of the participants, including the median, mean, minimum, and maximum values for laboratory parameters, are summarized in Table 1.

Correlations Between Vitamin D Levels and Laboratory Parameters

The relationships between vitamin D levels and biochemical parameters were evaluated using Spearman correlation analysis. A significant positive correlation was found between vitamin D levels and LDL-C ($\rho = 0.198$, $p = 0.0011$) and HDL-C ($\rho = 0.169$, $p = 0.0055$). Figures 2, 3, and 4 illustrate key findings related to vitamin D and lipid metabolism. Figure 2 demonstrates the positive correlation between serum vitamin D levels and LDL-C. Figure 3 depicts the distribution of HDL-C levels among the three vitamin D groups, while Figure 4 presents the mean HDL-C and LDL-C levels across these groups. Conversely, a significant negative correlation was observed between vitamin D levels and neutrophil counts ($\rho = -0.133$, $p = 0.030$). Other biochemical and hematological parameters, including triglycerides, glucose, lymphocyte count, monocyte count, and platelet

count, did not show statistically significant correlations with vitamin D levels ($p > 0.05$). Detailed correlation coefficients and p-values are presented in Table 2.

Further analyses of vitamin D levels with derived ratios, such as the triglyceride/glucose ratio, triglyceride/HDL-C ratio, triglyceride/monocyte ratio, monocyte/HDL-C ratio, neutrophil/lymphocyte ratio, and platelet/lymphocyte ratio, were conducted. Statistically significant positive correlations were observed only for the triglyceride/monocyte ratio ($\rho = 0.160$, $p = 0.0088$), while significant negative correlations were found for the monocyte/HDL-C ratio ($\rho = -0.203$, $p = 0.00083$). No significant correlations were identified for the other ratios ($p > 0.05$). The full results of these analyses are displayed in Table 3.

Group Comparisons Based on Vitamin D Levels

Participants were categorized into three groups based on their serum vitamin D levels:

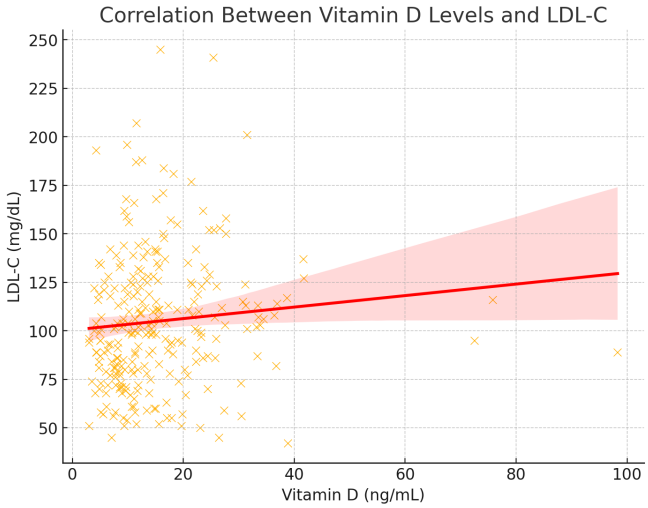
- Group 1: Deficient-insufficient (≤19 ng/mL, n = 198)
- Group 2: Sufficient (20–29 ng/mL, n = 46)
- Group 3: Normal (≥30 ng/mL, n = 23).

Differences in biochemical and hematological parameters, including LDL-C, HDL-C, triglycerides, glucose, neutrophil, lymphocyte, monocyte, and platelet counts, were analyzed among the groups using the Kruskal-Wallis test. A statistically significant difference among the groups was observed only for HDL-C levels ($p = 0.0022$). Figure 1 illustrates the distribution of HDL-C levels across the three vitamin D groups. Pairwise comparisons with the Mann-Whitney U test revealed that HDL-C levels in Group 1 were significantly lower than those in both Group 2 ($p = 0.0151$) and Group 3 ($p = 0.0046$), while no significant difference was found between Group 2 and Group 3 ($p = 0.449$).

Analysis of Derived Ratios Among Vitamin D Groups

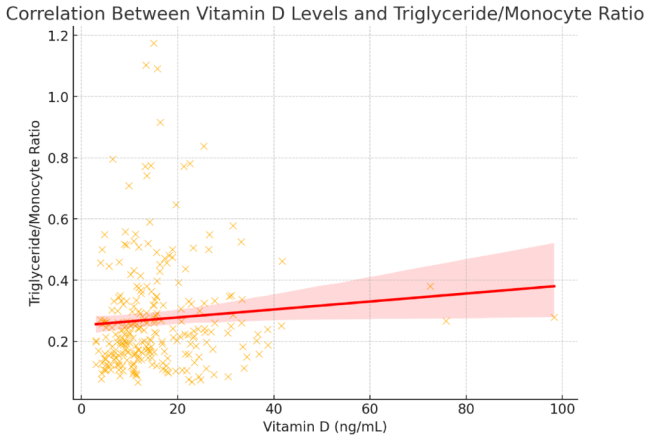
The derived ratios were also analyzed among the three vitamin D groups using the Kruskal-Wallis H test. A statistically significant difference was found only for the monocyte/HDL-C ratio ($p = 0.0013$). Post-hoc analyses using the Mann-Whitney U test indicated that the monocyte/HDL-C ratio was significantly higher in Group 1 compared to Group 3 ($p = 0.0018$) and moderately higher in Group 1 compared to Group 2 ($p = 0.0217$). No significant difference was found between Group 2 and Group 3 ($p = 0.2398$).

Figure 1: Correlation Between Vitamin D Levels and LDL-C



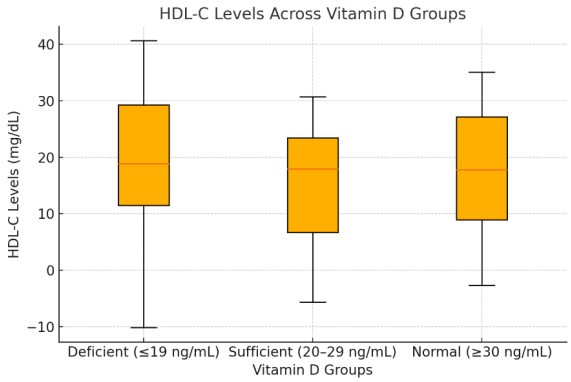
Scatter plot demonstrating the relationship between serum Vitamin D levels (ng/mL) and LDL-C levels (mg/dL).

Figure 2: Correlation Between Vitamin D Levels and Triglyceride/Monocyte Ratio



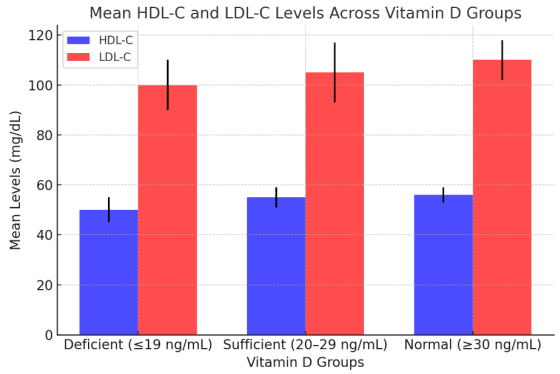
Scatter plot showing the correlation between serum Vitamin D levels (ng/mL) and the Triglyceride/Monocyte ratio.

Figure 3: HDL-C Levels Across Vitamin D Groups



A boxplot comparing HDL-C levels among the three Vitamin D groups.

Figure 4: Mean HDL-C and LDL-C Levels Across Vitamin D Groups



A bar chart comparing the mean HDL-C and LDL-C levels across Vitamin D groups.

Discussion

In this study, we explored the associations between vitamin D levels and various biochemical, hematological, and metabolic-inflammatory parameters in a healthy adult population. Our findings revealed significant positive correlations between vitamin D levels and both HDL-C and LDL-C, as well as a significant negative correlation with neutrophil counts. Additionally, among the derived metabolic-inflammatory ratios, a significant positive correlation was observed with the triglyceride/monocyte ratio and a significant negative correlation with the monocyte/HDL-C ratio. These findings provide novel insights into the broader metabolic and inflammatory effects of vitamin D in individuals without chronic disease or overt inflammatory conditions.

Our results align with previous studies suggesting a close relationship between vitamin D and lipid metabolism. Vitamin D has been shown to positively influence lipid profiles by improving HDL-C levels and reducing triglycerides and total cholesterol in certain populations [17,18]. In our study, the observed positive correlation between vitamin D levels and HDL-C is consistent with its known cardioprotective effects. HDL-C plays a critical role in reverse cholesterol transport, reducing the risk of atherosclerosis. The inter-group analysis further demonstrated that HDL-C levels were significantly lower in individuals with deficient vitamin D levels compared to those with sufficient or normal levels.

Interestingly, a positive correlation between vitamin D and LDL-C was also detected, which is contrary to most of the existing literature. While LDL-C is generally considered atherogenic, it is important to note that this relationship may reflect indirect effects of vitamin D on lipid metabolism, potentially mediated by dietary patterns, adipose tissue activity, or hepatic function [19-20]. Further studies are needed to clarify this association and its potential clinical implications.

Vitamin D is well-known for its immunomodulatory and anti-inflammatory properties, which are mediated through its effects on both innate and adaptive immune cells [21].

Studies have demonstrated that vitamin D inhibits the production of pro-inflammatory cytokines, such as TNF-α, IL-1β, and IL-6, while enhancing anti-inflammatory cytokines like IL-10 [22].In our study, we observed a significant negative correlation between vitamin D levels and neutrophil counts, supporting the potential role of vitamin D in reducing systemic inflammation. However, no significant correlations were found with the neutrophil/lymphocyte ratio (NLR) or platelet/lymphocyte ratio (PLR), which are established markers of inflammation. This may be due to the exclusion of individuals with acute or chronic inflammatory conditions, thereby limiting the variability in these parameters.

The absence of significant findings in the inflammatory ratios could also be explained by the low expression of

vitamin D receptors (VDR) in resting immune cells. Vitamin D primarily exerts its effects on activated immune cells, such as macrophages and T cells, where VDR expression is upregulated [23]. As our study population consisted of healthy individuals, the lack of immune activation may have contributed to these findings. Furthermore, the retrospective design and limited sample size of our study might have reduced the statistical power to detect subtle differences.

Insulin resistance is closely linked to lipid metabolism and systemic inflammation, and vitamin D is believed to play a key role in modulating both processes [24]. Vitamin D enhances insulin sensitivity by improving pancreatic beta cell function, increasing glucose uptake in peripheral tissues, and reducing systemic inflammation [25]. In our study, the triglyceride/monocyte ratio, which has been suggested as a potential marker of insulin resistance, showed a significant positive correlation with vitamin D levels. While previous studies have demonstrated a relationship between vitamin D and insulin resistance, the triglyceride/monocyte ratio is a relatively new marker in this context. Our findings highlight the need for further research to better understand the clinical utility of this ratio in assessing metabolic health.

Our findings underscore the multifaceted roles of vitamin D in lipid metabolism, systemic inflammation, and potentially insulin resistance. The positive correlation between vitamin D and HDL-C highlights its potential cardioprotective effects, while the observed associations with neutrophil counts and metabolic-inflammatory ratios suggest broader immunometabolic effects. However, given the retrospective design and small sample size of our study, caution is warranted when interpreting these findings.

Table 1: Baseline Characteristics of the Study Population

Parameter	Median (IQR)	Mean ± SD	Min–Max
Age (years)	35 (28–43)	35.1 ± 10.1	18–63
Vitamin D (ng/mL)	12.6 (8.2–19.4)	15.47 ± 10.4	3–98.3
B12 (pg/mL)	315 (250–380)	335.9 ± 118.3	100–871
Folate (ng/mL)	6.5 (4.9–8.2)	7.2 ± 2.5	1.6–20
HDL-C (mg/dL)	53 (45–61)	54.9 ± 13.3	5.2–107
Triglycerides (mg/dL)	91 (70–110)	107.6 ± 49.1	33–395
Glucose (mg/dL)	91 (87–96)	92.4 ± 8.2	64–123
Neutrophils (×10³/µL)	3.88 (3.1–4.6)	4.07 ± 1.2	1.37–9.39
Lymphocytes (×10³/µL)	2.15 (1.8–2.5)	2.24 ± 0.6	0.73–4.45
Monocytes (×10³/µL)	0.4 (0.3–0.5)	0.4 ± 0.1	0.2–0.8
Platelets (×10³/µL)	265 (220–310)	268.8 ± 60.2	136–572

Table 2: Correlations Between Vitamin D Levels and Biochemical Parameters

Blood Parameters	Correlation Coefficient (ρ)	p-value
LDL-C	0.198	0.0011
HDL-C	0.169	0.0055
Triglycerides	0.091	0.136
Glucose	0.037	0.549
Neutrophils	-0.133	0.03
Lymphocytes	-0.075	0.222
Monocytes	-0.115	0.061
Platelets	-0.108	0.078

Table 3: Correlations Between Vitamin D Levels and Derived Ratios

Ratio	Correlation Coefficient (ρ)	p-value
Triglyceride/Glucose	0.0885	0.149
Triglyceride/HDL-C	0.0051	0.934
Triglyceride/Monocyte	0.160	0.0088
Monocyte/HDL-C	-0.203	0.00083
Neutrophil/Lymphocyte	-0.0567	0.356
Platelet/Lymphocyte	-0.0118	0.848

Future studies with larger, multicenter cohorts and prospective designs are needed to validate these observations. Additionally, mechanistic studies exploring the molecular pathways linking vitamin D to lipid metabolism and inflammation could provide valuable insights into its therapeutic potential.

There are several limitations to our study. First, its retrospective nature may introduce selection bias, as data were collected from patient records, and the study population was limited to individuals presenting with fatigue. Second, the relatively small sample size, particularly in the normal vitamin D group, may have limited the statistical power to detect significant differences. Third, we did not account for potential confounding factors such as dietary intake, physical activity, or seasonal variation in vitamin D levels, which could influence the observed associations. Additionally, data on BMI, visceral adiposity, and body composition (e.g., Tanita measurements) were not available due to the retrospective design. These parameters could provide further insight into the relationship between vitamin D and systemic inflammation.

Furthermore, the study did not include subgroup analyses based on sex or menopausal status, which may influence lipid parameters. Future research should consider these factors for a more comprehensive analysis. Another limitation is that recent infection history, medication use, or physical activity levels, which are known to affect hematological and lipid parameters, were not assessed. These factors should be taken into account in prospective studies.

Finally, while we excluded individuals with overt inflammatory or infectious conditions, subclinical inflammation or other unmeasured confounders may have influenced the results. Additionally, the categorization of vitamin D levels was based on a cutoff of 30 ng/mL, whereas recent literature defines sufficiency at ≥20 ng/mL. This difference may have influenced the statistical distribution of the results. Future studies should consider this updated classification to enhance comparability with recent findings.

Conclusion

Our study demonstrates significant associations between vitamin D levels and lipid metabolism, as evidenced by its positive correlation with HDL-C, and with systemic inflammation, as indicated by its negative correlation with neutrophil counts. While the absence of significant findings in inflammatory ratios may reflect the healthy status of our population, the observed correlations suggest that vitamin D plays a regulatory role in both metabolic and inflammatory pathways. However, further large-scale, prospective studies are needed to confirm these findings and clarify their clinical implications.

Declarations

Ethics approval and consent to participate

Due to the retrospective nature of this study, informed consent was not obtained from the patients. This study was approved by Health Sciences University Umraniye Training and Research Hospital Clinical Research Ethics Committee, which is consistent with the 1964 Declaration of Helsinki.

Consent for publication

Not applicable

Availability of data and material

The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request. Due to privacy concerns and ethical

restrictions, the data are not publicly available. Any requests for data will be reviewed by the study's ethics committee to ensure compliance with ethical standards.

Competing interests

The authors declare that they have no competing interests.

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