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Effect of Vitamin D3 Supplementation on Interferon-Gamma Levels in Children with Nephrotic Syndrome

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KEYWORDS

Nephrotic syndrome; Vitamin D3; Interferon-gamma; Pediatric patients; Immune regulation

ABSTRACT:

Introduction: Nephrotic syndrome (NS) in children commonly leads to vitamin D deficiency through urinary loss of vitamin D–binding protein and albumin. Vitamin D regulates cytokine production, including interferon-gamma (IFN-γ), a key Th1 pro-inflammatory cytokine in NS immune dysregulation. Evidence on vitamin D supplementation's effect on IFN-γ levels in pediatric NS remains limited.

Objective: This study aimed to evaluate the effect of vitamin D3 supplementation on interferon-gamma levels in children with nephrotic syndrome.

Methods: A double-blind randomized controlled trial was conducted in children aged 1–18 years with nephrotic syndrome at two hospitals in Makassar, Indonesia. Participants received standard therapy with vitamin D3 supplementation of 400 IU/day or 1000 IU/day for four weeks. Serum 25-hydroxyvitamin D [25(OH)D] and interferon-gamma levels were measured before and after supplementation. Data analysis used Mann–Whitney and Wilcoxon tests ($p < 0.05$).

Results: A total of 59 participants completed the study (30 in the 400 IU group and 29 in the 1000 IU group). Baseline IFN-γ levels were comparable between groups. After four weeks of supplementation, serum vitamin D levels increased significantly in both groups ($p < 0.05$), although most participants remained below the normal range. The median increase in vitamin D levels was 4.7 ng/mL in the 400 IU group and 5.1 ng/mL in the 1000 IU group. Changes in IFN-γ levels were not statistically significant between groups.

Conclusion: Vitamin D3 supplementation improved serum vitamin D levels in children with nephrotic syndrome but did not significantly affect interferon-gamma levels within four weeks of intervention. Higher doses or longer supplementation may be required to achieve meaningful immunomodulatory effects.

1. Introduction

Nephrotic syndrome (NS) is a glomerular disorder characterized by massive proteinuria, hypoalbuminemia (<30 g/L), hyperlipidemia, and edema. Idiopathic nephrotic syndrome is the most common form in children and typically occurs between the ages of 2 and 5 years [1–3]. The incidence of pediatric NS is estimated at 12–16 cases per 100,000 children globally, while in Indonesia the annual incidence is approximately 6 cases per 100,000 children under 14 years of age [1,4].

Vitamin D deficiency is frequently observed in patients with NS. The relationship is considered bidirectional: vitamin D deficiency may contribute to podocyte injury, while nephrotic syndrome itself can cause vitamin D loss through urinary excretion of vitamin D–binding protein [5,6]. Several studies also suggest that vitamin D influences treatment response and steroid resistance through immunomodulatory mechanisms [7,8].

The pathogenesis of NS involves immune dysregulation and cytokine imbalance [9]. Interferon-gamma (IFN-γ), a key pro-inflammatory cytokine produced by Th1 cells, plays an important role in cellular immune responses. Previous studies have shown that IFN-γ levels are significantly lower during the active phase of NS compared with remission [10].

Vitamin D acts as an immunomodulator through the vitamin D receptor (VDR) and may regulate cytokine production, including IFN-γ [11]. However, evidence

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regarding the effect of vitamin D supplementation on IFN-γ levels in pediatric nephrotic syndrome remains limited. Therefore, this study aimed to evaluate the effect of vitamin D3 supplementation on interferon-gamma levels in children with nephrotic syndrome.

2. Methods

Study Design

This study was conducted as a double-blind randomized controlled trial (RCT) to evaluate the effect of vitamin D3 supplementation on interferon-gamma (IFN-γ) levels in children with nephrotic syndrome.

Study Setting and Period

The study was conducted at Dr. Wahidin Sudirohusodo Hospital and Jaury Jusuf Putera Academic Hospital, Makassar, Indonesia. Laboratory analyses were performed at the Research Laboratory of Hasanuddin University Hospital. Patient recruitment and data collection were carried out between August 2025 and January 2026.

Study Population and Sampling

The target population of this study consisted of pediatric patients diagnosed with nephrotic syndrome. The accessible population included children aged 1–18 years with nephrotic syndrome who were treated at Dr. Wahidin Sudirohusodo Hospital and Jaury Jusuf Putera Academic Hospital, Makassar, during the study period. The required sample size was determined based on previous studies, with a significance level of 5% and statistical power of 80%. The minimum sample size obtained was 18 participants in each group. To account for a potential 10% dropout rate, the final sample size was increased to 20 participants per group, resulting in a total of 40 participants included in the study.

Participants who met the eligibility criteria were recruited using a consecutive sampling technique. Eligible subjects were then randomly assigned into two groups using a double-blind allocation method. The first group received standard therapy for nephrotic syndrome along with vitamin D3 supplementation at a dose of 400 IU/day, while the second group received standard therapy combined with vitamin D3 supplementation at a dose of 1000 IU/day. Both participants and investigators were blinded to the treatment allocation throughout the study period.

Eligibility Criteria

Participants were eligible for inclusion if they were children aged 1–18 years diagnosed with nephrotic syndrome and received treatment at Dr. Wahidin Sudirohusodo Hospital or Jaury Jusuf Putera Academic Hospital, Makassar. Written informed consent was obtained from the parents or legal guardians prior to participation in the study. Patients were excluded if they had a history of malignancy, liver dysfunction, or had received daily vitamin D supplementation for three consecutive months prior to enrollment. Participants were considered dropouts if they failed to take vitamin D supplementation for three consecutive days, voluntarily withdrew from the study, or died before completion of the study period.

Data Collection

Clinical and demographic data were collected for all participants, including age, sex, body weight, height, and nutritional status. Anthropometric measurements were performed to determine nutritional status according to WHO and NCHS standards. Laboratory parameters were also recorded, including serum albumin levels, urinary protein levels, serum 25-hydroxyvitamin D [25(OH)D] levels, and serum IFN-γ levels.

Laboratory Analysis

Blood samples (3 mL) were collected from peripheral veins using sterile disposable syringes. Serum samples were separated by centrifugation within 30 minutes after collection and stored at 2–8°C before analysis.

Serum 25-hydroxyvitamin D [25(OH)D] levels were measured using chemiluminescence immunoassay (CLIA). Serum IFN-γ levels were measured using the BioLegend MAX™ Human IFN-γ ELISA Kit based on the sandwich enzyme-linked immunosorbent assay (ELISA) technique according to the manufacturer's protocol. Samples and standards were analyzed in duplicate using a 96-well microplate system.

Ethical Considerations

Ethical approval for this study was obtained from the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University (Protocol No. UH25090721; Ethical Approval No. 796/UN4.6.4.5.31/PP36/2025, issued on 15 October 2025). The study was conducted in accordance with the ethical standards of biomedical research involving human subjects. Prior to participation, written informed consent was obtained from the parents or legal guardians of all participants. Participants were informed about the study procedures and their right to withdraw from the study at any time without affecting their medical care.

Statistical Analysis

Data were analyzed using SPSS version 27. Descriptive statistics were used to summarize baseline characteristics. Continuous variables were expressed as

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mean ± standard deviation or median (interquartile range) depending on data distribution.

Normality was assessed using the Kolmogorov–Smirnov test. Differences between groups were analyzed using the independent t-test or Mann–Whitney U test. Within-group comparisons before and after intervention were evaluated using the paired t-test or Wilcoxon signed-rank test. Correlations between vitamin D levels and IFN-γ were analyzed using Pearson or Spearman correlation tests. A p-value < 0.05 was considered statistically significant.

3. Results

Study Participants

This clinical trial evaluated the effect of vitamin D supplementation on interferon-gamma levels in children with nephrotic syndrome. A total of 66 pediatric patients aged 1–18 years with nephrotic syndrome from the outpatient and inpatient departments of Dr. Wahidin Sudirohusodo Hospital and Jaury Jusuf Putera Academic Hospital, Makassar**,** met the inclusion criteria and were enrolled in the study. During the intervention period, 7 participants dropped out, resulting in 59 patients who completed the study protocol. The participants were divided into two groups based on the intervention: 30 patients received vitamin D3 supplementation of 400 IU/day**,** while 29 patients received vitamin D3 supplementation of 1,000 IU/day.

The characteristics of the study participants are presented in Table 1. There was a significant difference between Group I and Group II in terms of nutritional status ($p < 0.05$). However, no significant differences were observed between the groups with respect to age, sex, baseline vitamin D status, post-intervention vitamin D status, and remission status ($p > 0.05$).

Comparison of Interferon-Gamma and Vitamin D Levels Before Vitamin D3 Supplementation

Serum interferon-gamma and vitamin D levels were measured prior to vitamin D3 supplementation in the 400 IU and 1000 IU groups, as presented in Table 2. Data normality was assessed using the Kolmogorov–Smirnov test, which showed that the distributions of pretest, posttest, and change values for both vitamin D ($p = 0.023$; $p = 0.001$; $p = 0.019$) and interferon-gamma ($p < 0.001$) were not normally distributed.

Before supplementation, the median vitamin D level was 10.2 ng/mL in the 1000 IU group and 8.2 ng/mL in the 400 IU group, with no statistically significant difference ($p = 0.200$). Similarly, the median interferon-gamma level was 8.41 pg/mL in the 1000 IU group and 8.59 pg/mL in the 400 IU group, also showing no significant difference between groups ($p = 0.559$).

Comparison of Interferon-Gamma and Vitamin D Levels After Vitamin D3 Supplementation

Serum interferon-gamma and vitamin D levels after vitamin D3 supplementation in the 400 IU and 1000 IU groups are presented in Table 3. After supplementation, the median vitamin D level was 14.6 ng/mL in the 1000 IU group and 12.9 ng/mL in the 400 IU group, with no statistically significant difference between the groups ($p = 0.347$). Similarly, the median interferon-gamma level was 10.02 pg/mL in the 1000 IU group and 9.35 pg/mL in the 400 IU group, also showing no significant difference ($p = 0.534$).

Changes in Interferon-Gamma and Vitamin D Levels Before and After Vitamin D3 Supplementation

The changes in serum interferon-gamma and vitamin D levels before and after vitamin D3 supplementation in the 400 IU and 1000 IU groups are presented in Table 4. The median increase in vitamin D levels was 5.1 ng/mL in the 1000 IU group and 4.7 ng/mL in the 400 IU group, with no significant difference between groups ($p = 0.903$).

For interferon-gamma, the 1000 IU group showed a median increase of 0.57 pg/mL, whereas the 400 IU group showed a median decrease of −1.43 pg/mL. However, the difference in changes between the two groups was not statistically significant ($p = 0.324$).

Based on Table 5, the 1000 IU/day vitamin D3 group showed a median increase in interferon-gamma levels of 0.57 pg/mL, but the change was not statistically significant ($p = 0.689$). In contrast, the 400 IU/day group showed a median decrease in interferon-gamma levels of −1.43 pg/mL, which was also not statistically significant ($p = 0.237$).

Based on Table 6, both groups demonstrated an increase in vitamin D levels. In the 1000 IU/day group, the median increase was 5.1 ng/mL, showing a statistically significant change after intervention ($p = 0.018$). Similarly, the 400 IU/day group showed a median increase of 4.7 ng/mL, which was also statistically significant ($p = 0.001$).

4. Discussion

This randomized double-blind clinical trial evaluated the effect of vitamin D3 supplementation on IFN-γ levels in children with idiopathic nephrotic syndrome (INS). The baseline characteristics of the study population showed a predominance of male patients (64.4%), which is consistent with previous studies reporting that boys have approximately 1.8–2 times higher risk of developing INS compared with girls due to genetic variations affecting

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immune pathways [12]. The majority of participants were in the school-age group (5–12 years), which may reflect referral patterns in tertiary hospitals where relapsing or chronic cases are more frequently encountered. Importantly, age and sex distribution were comparable between groups, supporting the internal validity of the study.

A significant difference was observed in nutritional status between groups. Malnutrition is frequently encountered in children with nephrotic syndrome and may influence vitamin D metabolism. Vitamin D circulates bound to albumin and vitamin D binding protein (VDBP); therefore, hypoalbuminemia and protein loss may reduce vitamin D bioavailability [13]. Conversely, obesity has also been associated with lower circulating vitamin D levels due to sequestration in adipose tissue, further reducing its bioavailability [14]. These findings highlight the complex interaction between nutritional status and vitamin D metabolism in pediatric nephrotic syndrome.

At baseline, most participants had vitamin D deficiency or severe deficiency, which is consistent with previous reports indicating that vitamin D deficiency is highly prevalent in nephrotic syndrome. Massive proteinuria results in urinary loss of VDBP and albumin, leading to decreased circulating 25(OH)D levels [15,16]. In addition, impaired tubular reabsorption of the vitamin D–VDBP complex due to dysfunction of the megalin–cubilin receptor system further contributes to vitamin D depletion [17]. These mechanisms explain why children with active nephrotic syndrome frequently present with severe vitamin D deficiency.

Following supplementation, both groups demonstrated a significant increase in serum vitamin D levels. However, the magnitude of increase did not differ significantly between the 400 IU and 1000 IU groups. Despite this improvement, post-intervention median vitamin D levels remained below the threshold for sufficiency (>30 ng/mL), suggesting that these doses may be insufficient for children with active nephrotic syndrome. Previous studies have suggested that higher doses of vitamin D supplementation may be required to overcome persistent urinary losses and achieve optimal serum levels in nephrotic patients [18,19]. Long-term corticosteroid therapy may further exacerbate vitamin D deficiency by inducing CYP24A1-mediated catabolism of vitamin D metabolites [20].

In terms of immunological response, baseline IFN-γ levels were comparable between groups, indicating similar immune status prior to intervention. IFN-γ is a key Th1-associated pro-inflammatory cytokine involved in immune dysregulation observed in nephrotic syndrome [21]. Vitamin D has been shown to exert immunomodulatory effects through activation of the vitamin D receptor (VDR), which suppresses Th1 cytokine production, including IFN-γ [22].

However, vitamin D supplementation for four weeks did not significantly alter IFN-γ levels in either group. Several factors may explain this finding. First, all patients received corticosteroids as part of standard therapy, which strongly suppress adaptive immune responses and may mask the immunomodulatory effects of vitamin D supplementation [22]. Second, persistent urinary loss of VDBP in active nephrotic syndrome may prevent serum vitamin D levels from reaching the threshold required to exert systemic immunomodulatory effects [19]. Finally, the relatively short duration of supplementation may not be sufficient to induce measurable changes in cytokine profiles.

Although the difference in remission rates between groups was not statistically significant, the 1000 IU group showed a numerically higher remission proportion. Previous studies have suggested that adequate vitamin D status may improve regulatory T-cell activity and reduce relapse frequency in children with nephrotic syndrome [23]. Therefore, longer supplementation duration or higher doses may be necessary to achieve meaningful clinical and immunological effects.

This study has several limitations. The relatively short intervention period of four weeks may not fully capture long-term immunological changes. The sample size was modest and derived from a single center, which may limit generalizability. In addition, differences in baseline nutritional status and the universal use of corticosteroids may have introduced confounding or masking effects on cytokine responses.

Nevertheless, this study has important strengths. The double-blind randomized controlled design provides a high level of evidence, and the study evaluates IFN-γ as an immunological biomarker that has rarely been investigated in relation to vitamin D supplementation in pediatric nephrotic syndrome.

5. Limitations

This study has several limitations. First, adjustments for potential confounding factors such as dietary intake and sunlight exposure were not performed. Second, a significant difference in baseline nutritional status between the two groups may have influenced the study outcomes. Finally, outcome measurements were conducted at a single post-intervention time point, namely four weeks after the intervention, which may limit the evaluation of longer-term effects.

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6. Conclusion

This study demonstrated that children with nephrotic syndrome had low baseline vitamin D levels prior to supplementation. Although interferon-gamma (IFN-γ) levels were comparable between groups at baseline, vitamin D deficiency was prevalent among the participants. Following four weeks of vitamin D3 supplementation, serum vitamin D levels increased significantly in both the 400 IU and 1000 IU groups; however, most post-intervention levels remained below the normal range.

No significant difference was observed in IFN-γ levels between the two supplementation doses after intervention. While the 1000 IU group showed numerically higher post-supplementation vitamin D levels and slightly lower IFN-γ levels compared with the 400 IU group, these differences were not statistically significant. Overall, vitamin D3 supplementation improved serum vitamin D status in children with nephrotic syndrome but did not significantly modify IFN-γ levels within the four-week intervention period.

7. Declarations

Availability of Data and Materials

The datasets used and analyzed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The author declares that there are no competing interests related to this study.

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Authors' Contributions

F.R. contributed to conceptualization, data curation, formal analysis, investigation, methodology, and writing—original draft preparation. J. contributed to conceptualization, supervision, methodology, validation, and writing—review and editing. S.A.L. contributed to investigation, methodology, data curation, and writing—review and editing. H.A. contributed to data curation, investigation, and validation. U. contributed to investigation, data interpretation, and manuscript review. M.M.A. contributed to supervision, validation, and critical review of the manuscript. All authors have read and approved the final manuscript.

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References

1. Nurtyas Y. The Correlation of Regulatory T (TReg) and Vitamin D3 in Pediatric Nephrotic Syndrome. *J Trop Life Sci*. 2018;8(1):43-47. doi:10.11594/jtls.08.01.08
2. Hassan I, Tiewsoh JBA, Ray P, et al. Changing Spectrum of Infections in Childhood Nephrotic Syndrome. *The Indian Journal of Pediatrics 2019 86:11*. 2019;86(11):1065-1065. doi:10.1007/s12098-019-03007-1
3. Tapia C, Bashir K. Nephrotic Syndrome. *StatPearls*. Published online 2026. Accessed March 7, 2026. https://pubmed.ncbi.nlm.nih.gov/29262216/
4. Chanchlani R, Parekh RS. Ethnic differences in childhood nephrotic syndrome. *Front Pediatr*. 2016;4(APR):180162. doi:10.3389/fped.2016.00039
5. Li YC. Renoprotective effects of vitamin D analogs. *Kidney Int*. 2010;78(2):134-139. doi:10.1038/ki.2009.175
6. Nakayama Y, Ueda S, Okuda S. Molecular mechanism underlying the renoprotective action of vitamin D. *Circ J*. 2014;78(3):599-600. doi:10.1253/circj.CJ-14-0058
7. Bennett MR, Pordal A, Haffner C, Pleasant L, Ma Q, Devarajan P. Urinary Vitamin D-Binding Protein as a Biomarker of Steroid-Resistant Nephrotic Syndrome. *Biomark Insights*. 2016;11:1. doi:10.4137/Bmi.s31633
8. Hackl A, Zed SEDA, Diefenhardt P, Binz-Lotter J, Ehren R, Weber LT. The role of the immune system in idiopathic nephrotic syndrome. *Mol Cell Pediatr*. 2021;8(1). doi:10.1186/s40348-021-00128-6
9. Ahmadian E, Rahbar Saadat Y, Dalir Abdolahinia E, et al. The Role of Cytokines in Nephrotic Syndrome. *Mediators Inflamm*. 2022;2022:6499668. doi:10.1155/2022/6499668

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www.jchr.org

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10. Kim SH, Park SJ, Han KH, et al. Pathogenesis of minimal change nephrotic syndrome: an immunological concept. *Korean J Pediatr*. 2016;59(5):205-211. doi:10.3345/kjp.2016.59.5.205

11. Ghaseminejad-Raeini A, Ghaderi A, Sharafi A, et al. Immunomodulatory actions of vitamin D in various immune-related disorders: a comprehensive review. *Front Immunol*. 2023;14:950465. doi:10.3389/fimmu.2023.950465

12. Seyezadeh A, Tohidi MR, Sheikhi S, et al. Clinical Features of Idiopathic Nephrotic Syndrome and Factors Associated with its Relapse in Children: A Population-Based Study. *Journal of Comprehensive Pediatrics 2020 11:4*. 2020;11(4):e108024. doi:10.5812/COMPREPED.108024

13. Jørgensen HS, Vervloet M, Cavalier E, et al. The role of nutritional vitamin D in chronic kidney disease–mineral and bone disorder in children and adults with chronic kidney disease, on dialysis, and after kidney transplantation—a European consensus statement. *Nephrology Dialysis Transplantation*. 2025;40(4):797-822. doi:10.1093/ndt/gfae293

14. Cominacini M, Fumaneri A, Ballerini L, Braggio M, Valenti MT, Dalle Carbonare L. Unraveling the Connection: Visceral Adipose Tissue and Vitamin D Levels in Obesity. *Nutrients 2023, Vol 15, Page 4259*. 2023;15(19):4259. doi:10.3390/nu15194259

15. Selewski DT, Chen A, Shatat IF, et al. Vitamin D in incident nephrotic syndrome: a Midwest Pediatric Nephrology Consortium study. *Pediatric Nephrology*. 2016;31(3):465-472. doi:10.1007/s00467-015-3236-x

16. Bennett MR, Pordal A, Haffner C, Pleasant L, Ma Q, Devarajan P. Urinary Vitamin D-Binding Protein as a Biomarker of Steroid-Resistant Nephrotic Syndrome. *Biomark Insights*. 2016;11:1. doi:10.4137/Bmi.s31633

17. Kaseda R, Hosojima M, Sato H, Saito A. Role of megalin and cubilin in the metabolism of vitamin D(3). *Ther Apher Dial*. 2011;15 Suppl 1(SUPPL.1):14-17. doi:10.1111/j.1744-9987.2011.00920.x

18. Kogon AJ, Ballester LS, Zee J, et al. Vitamin D Supplementation in Children and Young Adults with Persistent Proteinuria Secondary to Glomerular Disease. *Pediatr Nephrol*. 2022;38(3):749. doi:10.1007/s00467-022-05660-9

19. Selewski DT, Chen A, Shatat IF, et al. Vitamin D in incident nephrotic syndrome: a Midwest Pediatric Nephrology Consortium study. *Pediatric Nephrology*. 2016;31(3):465-472. doi:10.1007/s00467-015-3236-x

20. Nowacka A, Śniegocki M, Bożiłow D, Ziółkowska EA. CYP24A1 in Small Intestinal Vitamin D Metabolism and Clinical Implications. *Nutrients 2025, Vol 17, Page 3348*. 2025;17(21):3348. doi:10.3390/nu17213348

21. Hackl A, Zed SEDA, Diefenhardt P, Binz-Lotter J, Ehren R, Weber LT. The role of the immune system in idiopathic nephrotic syndrome. *Mol Cell Pediatr*. 2021;8(1):18. doi:10.1186/s40348-021-00128-6

22. Ahmadian E, Rahbar Saadat Y, Dalir Abdolahinia E, et al. The Role of Cytokines in Nephrotic Syndrome. *Mediators Inflamm*. 2022;2022:6499668. doi:10.1155/2022/6499668

23. Subandiyah K, Khusna HK. The effect of vitamin 25-(OH)-D3 on Glucocorticoid Receptor-β (GRβ) and 25-(OH)-D receptors in idiopathic nephrotic syndrome patients. *Pediatric Sciences Journal*. 2020;1(1):15-19. doi:10.51559/pedscij.v1i1.4

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TABLES

Table 1. Characteristics of Study Participants

Characteristic	Vitamin D3 400 IU (n=30)	Vitamin D3 1000 IU (n=29)	p-value
	n (%)	n (%)	
Sex			0.145
Male	22 (73.3)	16 (55.2)	
Female	8 (26.7)	13 (44.8)	
Age (years)			0.302
<5 years	2 (6.7)	5 (17.2)	
5–12 years	15 (50.0)	16 (55.2)	
>12 years	13 (43.3)	8 (27.6)	
Nutritional Status			0.002
Severely undernourished	2 (6.7)	0 (0)	
Underweight	1 (3.3)	9 (31.0)	
Normal	21 (70.0)	19 (65.5)	
Overweight	1 (3.4)	1 (3.4)	
Obese	5 (16.7)	0 (0)	
Vitamin D Status (Pretest)			0.522
Severe deficiency	10 (33.3)	8 (27.6)	
Deficiency	15 (50.0)	11 (37.9)	
Insufficiency	4 (13.3)	8 (27.6)	
Normal	1 (3.3)	2 (6.9)	
Vitamin D Status (Posttest)			0.783
Severe deficiency	4 (13.3)	5 (17.2)	
Deficiency	14 (46.7)	10 (34.5)	
Insufficiency	8 (26.7)	8 (27.6)	
Normal	4 (13.3)	6 (20.7)	
Remission			0.355
Yes	8 (26.7)	11 (37.9)	
No	22 (73.3)	18 (62.1)	

Table 2. Comparison of Interferon-Gamma and Vitamin D Levels Before Vitamin D3 Supplementation Between the 400 IU and 1000 IU Groups

Characteristics	Group I (Vitamin D3 400IU)	Group II (Vitamin D3 1000IU)	p-value
	Median (Min-Maks)	Median (Min-Maks)	
Vitamin D (ng/mL)	8.2 (0.5-31)	10.2 (1.4-32)	0.200
Interferon Gamma (pg/ml)	8.59 (1.7-498.7)	8.41 (1.2-529.7)	0.559

Mann–Whitney test; statistically significant if $p < 0.05$.

Table 3. Comparison of Interferon-Gamma and Vitamin D Levels After Vitamin D3 Supplementation Between the Group Receiving 400 IU Vitamin D3 and the Group Receiving 1000 IU Vitamin D3 Supplementation

Characteristics	Group I (Vitamin D3 400IU)	Group II (Vitamin D3 1000IU)	p-value
	Median (Min-Maks)	Median (Min-Maks)	
Vitamin D (ng/mL)	12.9 (2-54.9)	14.6 (1.2-42.5)	0.347

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Interferon Gamma (pg/ml)	9.35 (0.5-91)	10.02 (0.2-583.8)	0.534

Mann–Whitney test; statistically significant if $p < 0.05$.

Table 4. Comparison of Changes in Interferon-Gamma and Vitamin D Levels After Vitamin D3 Supplementation Between the 400 IU and 1000 IU Vitamin D3 Groups

Characteristics	**Group I (Vitamin D3 400IU)**	**Group II (Vitamin D3 1000IU)**	**p-value**
	Median (Min-Maks)	**Median (Min-Maks)**	
Vitamin D (ng/mL)	4.7 ((-9.2)-44.4)	5.1 ((-28.1)-29.4)	0.903
Interferon Gamma (pg/ml)	-1.43 ((-490.13)–86.31)	0.57 ((-524.32)- 517.79)	0.324

Mann–Whitney test; statistically significant if $p < 0.05$.

Table 5. Comparison of Changes in Interferon-Gamma Levels Before and After Vitamin D3 Supplementation Between the 1000 IU and 400 IU Vitamin D3 Groups

Characteristics	**Interferon-Gamma Pretest**	**Interferon-Gamma Posttest**	**Deviation**	**p-value**
	Median (Min-Max)	**Median (Min-Max)**	**Median (Min-Max)**	
Group I (Vitamin D3 400 IU)	8.59 (1.7-498.7)	9.35 (0.5-91.0)	-1.43 ((-490.13)–86.31)	0.237
Group II (Vitamin D3 1000 IU)	8.41 (1.2-529.7)	10.02 (0.2-583.8)	0.57 ((-524.32)- 517.79)	0.689

Wilcoxon test; statistically significant if $p < 0.05$.

Table 6. Comparison of Changes in Serum Vitamin D Levels Before and After Vitamin D3 Supplementation Between the 400 IU and 1000 IU Vitamin D3 Groups

Characteristics	**Vitamin D Pretest**	**Vitamin D Posttest**	**Deviation**	**p-value**
	Median (Min-Max)	**Median (Min-Max)**	**Median (Min-Max)**	
Group I (Vitamin D3 400 IU)	8,295 (0,5-31)	12,9 (2-54,9)	4,7 ((-9,2)–44,4)	0,001
Group II (Vitamin D3 1000 IU)	10,2 (1,4-32)	14,6 (1,2-42,5)	5,1 ((-28,1)- 29,4)	0,018

Wilcoxon test; statistically significant if $p < 0.05$.