



Effect of Vitamin D Replacement in Patient with Sub-clinical Hypothyroidism

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ARTICLE INFO

Keywords: Endocrine System, Hypothyroidism, Pakistan, Thyroid Diseases, Vitamin D.

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Declaration

Authors' Contribution

All authors equally contributed to the study and approved the final manuscript

Conflict of Interest: No conflict of interest.

Funding: No funding received by the authors.

Article History

Received: 03-09-2025 Revised: 01-10-2025
Accepted: 07-10-2025 Published: 20-10-2025

ABSTRACT

Background: Subclinical hypothyroidism defines elevated serum thyroid stimulating hormone with normal thyroxine in the circulation. While often symptomless, subclinical hypothyroidism can evolve towards overt disease and cause metabolic as well as cardiovascular complications. Deficiency in vitamin D is extremely common and has been implicated in defective thyroid physiology by virtue of its immunomodulatory action. It has been posited that supplementation might correct thyroidal as well as metabolic outcomes in individuals so affected. **Objective:** To evaluate the effect of vitamin D replacement on thyroid stimulating hormone levels in patients with subclinical hypothyroidism. **Study Design:** It was Quasi-experimental study. **Study Duration and Setting:** Conducted from June 2025 to August 2025 at PAF Hospital, Islamabad. **Methodology:** A total of 60 patients aged 18–60 years with subclinical hypothyroidism were included. Participants received oral vitamin D supplementation of 50,000 IU weekly for eight weeks. Serum thyroid stimulating hormone, thyroxine, and vitamin D levels were measured before and after intervention. **Results:** The mean age of participants was 38.5 ± 12.7 years, with female predominance (78.3%). Vitamin D supplementation significantly reduced mean thyroid stimulating hormone levels from 7.05 ± 1.25 to 3.44 ± 1.38 mIU/L ($p < 0.001$). Serum Vit D increased from 16.07 ± 5.02 to 37.08 ± 7.22 ng/mL ($p < 0.001$). **Conclusion:** Vitamin D replacement therapy effectively improves thyroid function in patients with subclinical hypothyroidism.

INTRODUCTION

Subclinical hypothyroidism is a thyroid condition marked by increased blood thyroid-stimulating hormone (TSH) levels, although the circulating levels of free thyroxine (T₄) and triiodothyronine (T₃) are maintained within normal physiological ranges.¹ It usually represents an early phase of thyroid gland dysfunction, and most cases occur due to autoimmune thyroiditis.² The manifestations are generally mild, and many individuals remain asymptomatic or demonstrate nonspecific features such as lethargy, weight gain, cold intolerance, or cognitive impairment.³ Although overt hypothyroidism has been established as a significant etiology of metabolic and cardiovascular disorders, subclinical hypothyroidism also influences dyslipidemia, endothelial function, and neuropsychiatric domains.⁴ Since this is a sub-clinical state, many patients remain undiagnosed, but over time it can progress to overt hypothyroidism, particularly in those with circulating thyroid autoantibodies.⁵

Vitamin D is a fat-soluble secosteroid hormone that plays a central role in calcium and phosphorus homeostasis and skeletal integrity.⁶ Beyond its classical action on bone mineralization, contemporary research has demonstrated its effects on immunomodulation,

inflammatory cascades, and endocrine regulation.⁷ Hypovitaminosis D is a prevalent condition globally, irrespective of geography or socioeconomic strata. It is associated with multiple endocrine disorders including diabetes mellitus, polycystic ovary syndrome, and thyroid pathologies.⁸ Emerging evidence demonstrates an association between hypovitaminosis D and autoimmune thyroid disease, wherein deficient vitamin D status augments immune reactivity and accelerates thyroidal autoimmunity, leading to functional compromise of the gland.⁹

Correction of hypovitaminosis D in patients with sub-clinical hypothyroidism has received considerable scientific focus because of its potential benefits on thyroid physiology and metabolic regulation.¹⁰ Supplementation can decrease serum thyroid autoantibody titers, thereby lowering the risk of progression to overt hypothyroidism in individuals with autoimmune thyroiditis.¹¹ Moreover, restoration of adequate vitamin D status improves systemic metabolic parameters, particularly lipid profile and insulin sensitivity, which are frequently impaired in hypothyroid states.¹⁰ Controlled clinical trials have demonstrated that vitamin D replacement may reduce thyroid stimulating hormone concentrations and alleviate

patient-reported symptoms, although outcomes remain heterogeneous due to variations in dosage, treatment duration, and baseline nutritional condition.¹² In the study conducted by Pezeshki B. et al., the mean thyroid-stimulating hormone (TSH) concentration in patients with sub-clinical hypothyroidism decreased from 6.89 ± 1.48 mIU/L at baseline to 3.34 ± 1.35 mIU/L following vitamin D treatment.¹²

Vitamin D deficiency is very common in the Pakistani population owing to restricted sun exposure, traditional customs, and poor dietary intake. Concurrently, sub-clinical hypothyroidism is underdiagnosed and tends to advance towards overt disease with metabolic as well as cardiovascular morbidity. There is a lack of local data on the impact of vitamin D replacement therapy on thyroid function in sub-clinical hypothyroidism despite its substantial burden. Carrying out this research in Pakistan is thus imperative to provide indigenous evidence, inform clinical practice, and facilitate preventive approaches that are suited to the local population.

METHODOLOGY

The present investigation followed a quasi-experimental design and was carried out at PAF Hospital, Islamabad, from June to August 2025. Sixty individuals with biochemically confirmed subclinical hypothyroidism were recruited. The sample size was calculated with 95% confidence and 80% statistical power, based on prior evidence showing a reduction in mean thyroid-stimulating hormone (TSH) levels from 6.89 ± 1.48 mIU/L at baseline to 3.34 ± 1.35 mIU/L after vitamin D therapy. The study protocol received approval from the Institutional Review Board of PAF Hospital. Participants included male and female adults aged 18 to 60 years who demonstrated serum TSH concentrations between 5–10 mIU/L, with free thyroxine (T4) levels remaining within the normal reference range of 0.8–2.0 ng/dL. Vitamin D deficiency was defined as serum 25-hydroxyvitamin D levels below 30 ng/mL. Patients were excluded if they had overt hypothyroidism (TSH >10 mIU/L or reduced T4), pregnancy, autoimmune thyroid disease, significant cardiovascular, hepatic, or renal illness, or if they were receiving corticosteroids, levothyroxine, or vitamin D supplementation in the preceding year. Written informed consent was obtained before enrollment. A comprehensive history of thyroid-related symptoms, coexisting medical problems, and prior therapies was collected, followed by a physical assessment comprising vital signs and thyroid gland examination.

For biochemical assessment, venous samples were obtained under aseptic conditions after overnight fasting.

Table II

Effect of Vitamin D Replacement on TSH and Vitamin D Levels

Parameter	Time Point	Mean $\pm$ SD	Mean Difference	95% CI of Difference	t Value	p-value
TSH (mIU/L)	Baseline	7.05 ± 1.25	3.61	3.39 to 3.83	32.84	<0.001*
	After eight weeks	3.44 ± 1.38				
Vitamin D (ng/mL)	Baseline	16.07 ± 5.02	-21.01	-22.50 to -19.52	-28.24	<0.001*
	After eight weeks	37.08 ± 7.22				

* Paired-samples t-test

The correlation analysis identified several significant associations among the studied variables. Age was

At study entry, 5 mL of blood was drawn to determine serum TSH, free T4, and vitamin D levels. The same investigations were repeated at the end of the eight-week intervention period. Each patient was prescribed oral vitamin D at a dose of 50,000 IU once weekly for eight weeks. The primary endpoint was the change in TSH levels from baseline to post-treatment, while secondary outcomes included improvements in serum vitamin D status. The statistical analysis was conducted with SPSS version 26. Continuous data were presented as mean with standard deviation, whilst categorical data were represented as counts and percentages. The paired sample t-test was utilised to evaluate the differences between before and after the intervention data. Relationships among continuous variables were examined using Pearson's correlation analysis. A probability value under 0.05 was deemed statistically significant.

RESULTS

The study included 60 patients with a mean age of 38.50 ± 12.72 years and mean BMI of 27.53 ± 2.37 kg/m². The majority of participants were female (n=47, 78.3%) while males comprised 13 (21.7%) of the cohort. Regarding comorbidities, 10 patients (16.7%) had diabetes while 50 patients (83.3%) did not have diabetes (as shown in Table-I).

Table I

Patient Demographics

Demographics	Mean $\pm$ SD / n (%)
Age (years)	38.50 ± 12.72
BMI (kg/m ²)	27.53 ± 2.37
Gender	
Male n (%)	13 (21.7%)
Female n (%)	47 (78.3%)
Diabetes	
Yes n (%)	10 (16.7%)
No n (%)	50 (83.3%)

Vitamin D replacement therapy demonstrated significant effects on both TSH and Vitamin D levels over the eight-week intervention period. At baseline, the mean TSH level was 7.05 ± 1.25 mIU/L, which decreased significantly to 3.44 ± 1.38 mIU/L after eight weeks of treatment, showing a mean difference of 3.61 (95% CI: 3.39 to 3.83, $t=32.84$, $p<0.001$). Concurrently, mean Vitamin D levels increased substantially from 16.07 ± 5.02 ng/mL at baseline to 37.08 ± 7.22 ng/mL after eight weeks, with a mean difference of -21.01 (95% CI: -22.50 to -19.52, $t=-28.24$, $p<0.001$). Both changes were statistically significant as determined by paired-samples t-test (as shown in Table-II).

positively correlated with body mass index (BMI) ($r = 0.414$, $p = 0.001$), indicating that BMI increased with advancing age. A significant inverse relationship was

observed between BMI and post-treatment TSH levels ($r = -0.291$, $p = 0.024$), suggesting that higher BMI was associated with lower TSH values following vitamin D therapy. In contrast, age did not demonstrate significant correlations with post-treatment TSH ($r = -0.236$, $p = 0.069$) or post-treatment vitamin D levels ($r = -0.069$, $p = 0.602$). Similarly, BMI was not significantly related to post-treatment vitamin D concentrations ($r = -0.144$, $p = 0.272$). Post-treatment TSH and vitamin D levels also showed no significant correlation ($r = -0.098$, $p = 0.456$) (as shown in Table-III).

Table III

Correlation Matrix of Age, BMI, Post-Treatment TSH and Vitamin D Levels

Variable	Age	BMI	Post-Treatment TSH	Post-Treatment Vitamin D
Age	1	0.414* ($p=0.001$)	-0.236 ($p=0.069$)	-0.069 ($p=0.602$)
BMI	0.414* ($p=0.001$)	1	-0.291* ($p=0.024$)	-0.144 ($p=0.272$)
Post-Treatment TSH	-0.236 ($p=0.069$)	-0.291* ($p=0.024$)	1	-0.098 ($p=0.456$)
Post-Treatment Vitamin D	-0.069 ($p=0.602$)	-0.144 ($p=0.272$)	-0.098 ($p=0.456$)	1

*Significant ≤ 0.05 level

DISCUSSION

This study highlights that vitamin D supplementation exerts a notable positive effect on thyroid function in individuals with subclinical hypothyroidism. After eight weeks of therapy, mean serum TSH levels decreased markedly from 7.05 ± 1.25 mIU/L to 3.44 ± 1.38 mIU/L, demonstrating a clinically meaningful improvement. These results reinforce the concept that vitamin D is actively involved in thyroid regulation and that correction of vitamin D deficiency may serve as a beneficial therapeutic strategy in this population. The observed effect is likely mediated through vitamin D's immunoregulatory actions, which include downregulation of pro-inflammatory cytokines and attenuation of autoimmune processes directed against thyroid tissue—a common underlying mechanism in subclinical hypothyroidism. The concurrent rise in serum vitamin D concentration from 16.07 ± 5.02 ng/mL to 37.08 ± 7.22 ng/mL confirms both effective supplementation and restoration of sufficient vitamin D status, shifting patients from a deficient to a physiologically adequate range. This normalization provides additional evidence that the therapeutic regimen achieved the intended biochemical targets alongside measurable endocrine improvement.

The outcomes of this study are in strong agreement with previous research exploring the impact of vitamin D supplementation on thyroid function among individuals with subclinical hypothyroidism. The pronounced decline in TSH levels observed here—from 7.05 ± 1.25 mIU/L to 3.44 ± 1.38 mIU/L after eight weeks of treatment—closely parallels the findings of Pezeshki et al.¹² who documented a reduction from 6.89 ± 1.48 mIU/L to 3.34 ± 1.35 mIU/L following two months of weekly vitamin D doses of 50,000

IU in a cohort of 40 patients. The similarity in both baseline TSH values and post-treatment outcomes, despite differences in study design or population characteristics, reinforces the consistency of vitamin D's therapeutic benefit and suggests a reproducible treatment effect across diverse settings. In line with this, Safari et al.¹³ reported a 20.54% reduction in TSH after 12 weeks of supplementation in women with subclinical hypothyroidism, further supporting the notion that vitamin D contributes meaningfully to TSH regulation regardless of dosing schedules or treatment durations.

The increase in serum vitamin D concentrations in the present study—from 16.07 ± 5.02 ng/mL to 37.08 ± 7.22 ng/mL—also mirrors the results of Pezeshki et al.¹² who noted a rise from 15.98 ± 6.62 ng/mL to 37.68 ± 16.92 ng/mL. This demonstrates that the supplementation regimen not only achieved biochemical sufficiency but also produced the therapeutic range necessary for thyroid function improvement. Furthermore, the baseline deficiency in vitamin D identified in our participants is consistent with the high prevalence of vitamin D insufficiency described in earlier studies involving subclinical hypothyroidism. Rathi Roopavathy et al.¹⁴ reported significantly lower vitamin D levels in female patients with subclinical hypothyroidism (18.15 ± 8.61 ng/mL) compared with healthy controls (25.81 ± 10.85 ng/mL), findings that align closely with our baseline value of 16.07 ± 5.02 ng/mL. Similarly, Amer et al.¹⁵ reported mean serum vitamin D of 20.1 ± 5.0 nmol/L in affected patients, with a strong negative correlation between TSH and vitamin D ($r = -0.81$, $p < 0.0001$), reinforcing the inverse association between these two parameters. Elamawy et al.¹⁶ also demonstrated universal vitamin D deficiency among their subclinical hypothyroid cohort, with a median level of 14.6 ng/mL. However, they observed only a weak positive correlation with TSH ($r = 0.279$, $p = 0.036$), suggesting that although vitamin D deficiency is a consistent feature across populations, the strength and direction of correlation may vary due to genetic predispositions, environmental exposures such as sunlight, dietary patterns, and variability in disease severity at baseline.

The demographic characteristics of our study population, with a mean age of 38.50 ± 12.72 years, female predominance of 78.3%, and mean BMI of 27.53 ± 2.37 kg/m², are consistent with the epidemiological patterns observed in other studies investigating subclinical hypothyroidism. Copari-Vargas et al.¹⁷ reported a similar female predominance (61.4%) with a mean age of 45.8 ± 13.5 years and BMI of 28.7 ± 6.1 kg/m² in their Hashimoto's thyroiditis cohort, while Pezeshki et al.¹² included 35 females out of 40 participants (87.5%) with a mean age of 36.7 ± 13.5 years, demonstrating that subclinical hypothyroidism disproportionately affects middle-aged women with slightly elevated BMI. This demographic pattern reflects the increased susceptibility of women to autoimmune thyroid disorders due to hormonal influences, X-chromosome-linked immune genes, and higher prevalence of autoimmune conditions in females.

The inverse connection between BMI and post-treatment TSH levels identified in our study is a

noteworthy discovery that necessitates meticulous interpretation within the framework of previous literature. Fang et al.¹⁸ established that elevated vitamin D levels correlate with reduced BMI in individuals with type 2 diabetes, whereas Dulger et al.¹⁹ identified a weak negative correlation between vitamin D and BMI ($r = -0.166$, $p = 0.024$) in pregnant women; however, the interplay between BMI and thyroid function in response to vitamin D supplementation is intricate and multifaceted. The negative correlation we observed may reflect the fact that adipose tissue serves as a storage site for vitamin D, and individuals with higher BMI may have different vitamin D metabolism, with greater capacity for vitamin D storage and potentially more pronounced deficiency at baseline, leading to more dramatic improvements following supplementation. Safari et al.¹³ notably reported significant improvements in body composition following vitamin D supplementation, with decreased fat mass percentage and increased fat-free mass percentage, suggesting that vitamin D's effects extend beyond thyroid function to metabolic parameters that may modulate the relationship between BMI and TSH response.

Our findings revealed a significant positive association between age and body mass index ($r = 0.414$, $p = 0.001$), which aligns with the well-documented trend of progressive weight gain and metabolic alterations that accompany advancing age. Comparable results have been reported in previous studies. Wu et al.²⁰ observed that advancing age was linked with a higher likelihood of vitamin D sufficiency (OR = 1.05) and also demonstrated an association between hypothyroidism and older age (OR = 1.07). Appunni et al.²¹ utilising data from the NHANES survey, showed that patients with vitamin D insufficiency had significantly elevated odds of acquiring hypothyroidism, with modified odds ratios of 1.7 for intermediate deficit and 1.6 for marked deficiency. Dulger et al.¹⁹ found no significant correlation between thyroid function tests and vitamin D levels in first-trimester pregnant women, suggesting that the relationship may be influenced by the unique hormonal environment of pregnancy, where substantial physiological changes in thyroid function and vitamin D metabolism occur to support fetal development. Rajendiran et al.²³ similarly found no association between vitamin D deficiency and hypothyroidism in children with delayed anterior fontanelle closure, indicating that the vitamin D-thyroid relationship may differ across age groups and specific clinical contexts. These contrasting findings emphasize the importance of considering population-specific factors, including age, pregnancy status, baseline thyroid function,

severity of vitamin D deficiency, presence of autoimmune markers, and genetic variations in vitamin D and thyroid hormone receptors, when interpreting the relationship between vitamin D and thyroid function.

The present study has several limitations that warrant consideration when interpreting the findings. First, the single-center design limits the generalizability of our results to broader populations with different ethnic backgrounds, dietary patterns, and environmental factors that may influence vitamin D metabolism and thyroid function. The relatively small sample size of 60 patients may not have provided adequate statistical power to detect weaker associations or subgroup differences, particularly in stratified analyses by gender, age groups, or comorbidity status. The absence of a control group receiving placebo is a significant limitation, as it prevents us from definitively attributing the observed improvements in TSH levels solely to vitamin D supplementation rather than natural fluctuations in thyroid function, regression to the mean, or other concurrent lifestyle modifications. The short follow-up period of eight weeks may not be sufficient to assess the long-term sustainability of TSH reduction or to evaluate whether continued vitamin D supplementation is necessary to maintain therapeutic benefits.

CONCLUSION

Our investigation demonstrated that vitamin D replacement therapy exerts a beneficial impact on thyroid function in individuals diagnosed with subclinical hypothyroidism. This effect was particularly evident through the marked decline in serum thyroid-stimulating hormone (TSH) levels observed after the intervention, indicating an improvement in thyroid regulatory mechanisms. The eight-week treatment regimen resulted in appropriate serum vitamin D levels for most subjects, underscoring the need of sufficient vitamin D in regulating thyroid function. These findings indicate that vitamin D treatment not only rectifies insufficiency but also aids in restoring thyroid equilibrium, hence emphasising its potential as an adjuvant therapeutic approach in the treatment of subclinical hypothyroidism.

Acknowledgments

We express our heartfelt gratitude to the dedicated healthcare professionals in our unit for their meticulous attention to documentation accuracy and systematic organization of clinical information, which proved essential to the successful completion of this research endeavor.

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