Research Article

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Vitamin D Levels and Thyroid Function in Patients with Hyperthyroidism and Hypothyroidism

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Abstract

Background: *Vitamin D deficiency is a widespread global health issue, affecting over one billion individuals. The biologically active form, 1,25-dihydroxyvitamin D [1,25(OH)$_2$D], has been shown to regulate both innate and adaptive immune responses. Immune cells—including monocytes, macrophages, dendritic cells, and lymphocytes—express vitamin D receptors, suggesting a potential link between vitamin D status and autoimmune disorders, particularly thyroid diseases such as Graves' disease and Hashimoto's thyroiditis.*

Methods: *A prospective study was conducted at Arogyam Medical College and Hospital (AMCH), Roorkee, India, between March and July 2023. A total of 100 participants were enrolled. Venous blood samples were collected to assess serum vitamin D, triiodothyronine (T3), thyroxine (T4), and thyroid-stimulating hormone (TSH) levels using chemiluminescence immunoassay. Statistical analyses were performed using SPSS version 20.*

Results: *Hypothyroid patients exhibited significantly lower mean serum vitamin D levels (16.27 ± 10.50 ng/mL) compared to controls (32.64 ± 2.26 ng/mL). In contrast, hyperthyroid patients had mean vitamin D levels within the normal range (39.35 ± 7.50 ng/mL), with no statistically significant difference from controls. TSH levels were markedly elevated in hypothyroid patients (10.03 ± 5.60 mU/L) and significantly suppressed in hyperthyroid patients (0.23 ± 0.12 mU/L), compared to controls (3.16 ± 1.40 mU/L).*

Conclusion: *The findings suggest a significant association between vitamin D deficiency and hypothyroidism, indicating a possible role in the pathogenesis of autoimmune thyroid disorders. Further large-scale, longitudinal studies are warranted to determine the therapeutic potential of vitamin D supplementation in thyroid disease management.*

Keywords: Vitamin D Deficiency, Thyroid Dysfunction, Hypothyroidism, Hyperthyroidism, Autoimmune Thyroid Disease

1. Introduction

The thyroid gland is a butterfly-shaped endocrine organ located anterior to the trachea, consisting of two lateral lobes connected by an isthmus. It synthesizes and secretes the hormones thyroxine (T4) and triiodothyronine (T3), which are essential regulators of metabolic homeostasis and cellular function [1]. Thyroid hormones influence nearly all tissues, playing a central role in basal metabolic rate, thermogenesis, and energy metabolism [2]. Their production is tightly controlled by the hypothalamic–pituitary–thyroid (HPT) axis through a negative feedback mechanism involving thyrotropin-releasing hormone (TRH) and thyroid-stimulating hormone (TSH) [3]. Peripheral conversion of T4 to the biologically active T3 is mediated by iodothyronine deiodinases, whereas conversion to reverse T3 results in an inactive metabolite [4].

Vitamin D deficiency has emerged as a major global public health concern, affecting over one billion individuals worldwide [5]. Traditionally recognized for its role in calcium homeostasis and bone metabolism, vitamin D is now increasingly acknowledged as a pleiotropic steroid prohormone involved in immune regulation, cellular proliferation, and differentiation [6]. Endogenous synthesis in the skin following ultraviolet B (UVB) exposure represents the primary source of vitamin D, with smaller contributions from dietary intake [7]. Vitamin D undergoes hepatic conversion to 25-hydroxyvitamin D [25(OH)D], the principal circulating form, and is subsequently hydroxylated in the kidneys to its active form, 1,25-dihydroxyvitamin D [$1,25(OH)_2D$] [8]. The biological effects of vitamin D are mediated via the vitamin D receptor (VDR), a nuclear transcription factor expressed in multiple tissues [9].

Beyond skeletal functions, vitamin D plays a critical role in modulating both innate and adaptive immune responses [10]. Immune cells, including monocytes, macrophages, dendritic cells, and T and B lymphocytes, express VDR and respond to vitamin D signalling [11]. Consequently, vitamin D deficiency has been implicated in the pathogenesis of several autoimmune diseases, including type 1 diabetes mellitus, multiple sclerosis, rheumatoid arthritis, and inflammatory bowel disease [12-14]. Increasing evidence also suggests a potential association between vitamin D deficiency and autoimmune thyroid disorders such as Graves' disease and Hashimoto's thyroiditis [15].

Hypothyroidism, characterized by reduced thyroid hormone production, is a common endocrine disorder with a higher prevalence in females and increasing incidence with age [16]. Autoimmune thyroiditis remains the most frequent cause of primary hypothyroidism [17]. Serum TSH measurement is the most sensitive indicator of thyroid dysfunction, with elevated levels typically reflecting hypothyroid states [18]. Notably, both thyroid hormones and vitamin D exert their effects through structurally similar nuclear receptors, suggesting potential molecular interactions [19]. Furthermore, polymorphisms in the VDR gene have been associated with increased susceptibility to autoimmune thyroid diseases [20].

Emerging evidence indicates that patients with hypothyroidism frequently exhibit reduced serum vitamin D levels, potentially due to impaired intestinal absorption and altered metabolic activity [21]. Several studies have also demonstrated correlations between serum 25(OH)D levels and TSH concentrations in individuals with thyroid disorders [22]. Serum 25(OH)D is widely accepted as the most reliable biomarker of vitamin D status due to its relatively long half-life, whereas circulating $1,25(OH)_2D$ is less suitable for assessment because of its short half-life and tight physiological regulation [23].

Despite growing interest in the immunomodulatory role of vitamin D, the relationship between vitamin D deficiency and thyroid dysfunction remains incompletely understood. Given the rising prevalence of thyroid disorders and hypovitaminosis D, investigating this association is of significant clinical importance. Therefore, the present study aims to evaluate serum vitamin D levels in patients with thyroid disorders and to explore the relationship between vitamin D status and thyroid hormone parameters (T3, T4, and TSH). Understanding this interaction may provide insights into disease pathophysiology and support potential strategies for screening and therapeutic intervention.

2. Materials and Methods

2.1. Study Design and Setting

This cross-sectional observational study was conducted in the Department of Biochemistry at Arogyam Medical College and Hospital (AMCH), Roorkee, India. Participants were recruited from the Departments of Internal Medicine and Otorhinolaryngology (ENT). The study was carried out over an eight-month period from February to September 2023. A total of 100 participants presenting with clinical features suggestive of thyroid dysfunction were enrolled.

2.2. Study Population

Eligible participants included patients diagnosed with thyroid disorders (hypothyroidism or hyperthyroidism), including both newly diagnosed and those receiving treatment. Cases with overt as well as subclinical thyroid dysfunction were included. Demographic and clinical data, including age, sex, occupation, residence, comorbidities, and pregnancy status (where applicable), were recorded using a structured proforma.

2.2.1. Inclusion and Exclusion Criteria

Inclusion criteria:

(i) Patients diagnosed with hypothyroidism or hyperthyroidism
(ii) Patients under treatment or recently diagnosed
(iii) Individuals with overt or subclinical thyroid dysfunction

Exclusion criteria:

(i) History of major surgery
(ii) Malignancy
(iii) Hepatic or renal disease
(iv) Patients receiving interferon therapy
(v) Use of beta-blockers

2.3. Sample Collection and Processing

Approximately 5 mL of venous blood was collected aseptically from each participant using clot activator vacutainer tubes. Samples were allowed to clot at room temperature and subsequently centrifuged to separate serum, which was used for biochemical analysis.

2.4. Biochemical Analysis

Serum levels of thyroid-stimulating hormone (TSH), free triiodothyronine (fT3), free thyroxine (fT4), and 25-hydroxyvitamin D [25(OH)D] were measured using an automated chemiluminescence immunoassay system (ADVIA Centaur, Siemens Healthineers). All assays were performed according to the manufacturer's instructions, and appropriate quality control measures were maintained throughout the study.

TSH levels were determined using a two-site sandwich chemiluminescent immunoassay, where the emitted signal was directly proportional to analyte concentration. In contrast, fT3, fT4, and 25(OH)D were measured using competitive immunoassays, in which the signal intensity was inversely proportional to the concentration of the respective analyte in the sample.

2.5. Statistical Analysis

Data were analyzed using Statistical Package for the Social Sciences (SPSS) version 20.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Comparisons between groups were performed using appropriate statistical tests, with a p-value < 0.05 considered statistically significant.

2.6. Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of Arogyam Medical College and Hospital. Written informed consent was obtained from all participants prior to inclusion in the study, in accordance with the principles of the Declaration of Helsinki.

2.7. Statistical Analysis

All data were verified, coded, and entered into IBM SPSS Statistics version 20.0 (IBM Corp., Armonk, NY, USA) for analysis. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages.

Descriptive statistics were used to summarize baseline characteristics and biochemical parameters. Inferential analysis was performed using the independent Student's t-test to compare means between groups, and correlation analysis was applied to assess relationships between vitamin D levels and thyroid function parameters. A p-value < 0.05 was considered statistically significant.

To ensure data reliability and validity, standard operating procedures (SOPs) were strictly followed for all laboratory analyses. The ADVIA Centaur analyzer was routinely calibrated, and all assays were performed in accordance with manufacturer guidelines.

3. Results

3.1. Baseline Biochemical Characteristics

The biochemical profiles of the control, hypothyroid, and hyperthyroid groups are presented in Tables 1–3.

In the control group (Table 1), all measured parameters were within normal physiological ranges. The mean serum vitamin D level (32.64 ± 2.26 ng/mL; 95% CI: 31.99–33.29) indicates sufficient vitamin D status. Similarly, mean T3 (2.85 ± 0.24 ng/mL), T4 (1.28 ± 0.37 µg/dL), and TSH (3.17 ± 1.40 mU/L) values confirm euthyroid function. The relatively narrow confidence intervals reflect low variability and homogeneity within the control population.

Parameter	Minimum	Maximum	Mean ± SD	95% CI of Mean
Vitamin D (ng/mL)	30.00	36.76	32.64 ± 2.26	31.99–33.29
T3 (ng/mL)	2.59	3.31	2.85 ± 0.24	2.78–2.92
T4 (µg/dL)	0.92	1.66	1.28 ± 0.37	1.17–1.39
TSH (mU/L)	1.30	4.59	3.17 ± 1.40	2.77–3.57
Footnote: Values are expressed as mean ± SD. CI: confidence interval				

Table 1: Biochemical Parameters in Control Subjects (n = 50)

In hypothyroid subjects (Table 2), a marked reduction in serum vitamin D levels was observed (16.27 ± 10.50 ng/mL; 95% CI: 13.25–19.29), indicating prevalent hypovitaminosis D. Notably, the wide standard deviation and confidence interval suggest considerable inter-individual variability in vitamin D status. TSH levels were substantially elevated (10.03 ± 5.61 mU/L; 95% CI: 8.42–11.64), consistent with hypothyroid physiology. Although mean T3 (3.05 ± 0.68 ng/mL) and T4 (1.05 ± 0.24 µg/dL) values showed variation, these remained relatively less altered compared to TSH, indicating that TSH is a more sensitive marker of thyroid dysfunction in this group.

Parameter	Minimum	Maximum	Mean ± SD	95% CI of Mean
Vitamin D (ng/mL)	4.21	53.89	16.27 ± 10.50	13.25–19.29
T3 (ng/mL)	0.94	4.21	3.05 ± 0.68	2.86–3.24
T4 (µg/dL)	0.65	1.52	1.05 ± 0.24	0.98–1.12
TSH (mU/L)	5.66	24.43	10.03 ± 5.61	8.42–11.64

Table 2: Biochemical Parameters in Hypothyroid Subjects (n = 50)

In hyperthyroid subjects (Table 3), mean vitamin D levels (39.35 ± 7.50 ng/mL; 95% CI: 37.20–41.50) were within or above the normal range, suggesting no evidence of deficiency. Thyroid hormone levels were elevated, with mean T3 (3.92 ± 0.66 ng/mL) and T4 (1.82 ± 0.59 µg/dL), while TSH levels were markedly suppressed (0.23 ± 0.11 mU/L; 95% CI: 0.20–0.26), consistent with hyperthyroid status. Compared with the control group, greater variability was observed in hormone levels, particularly for T4.

Parameter	Minimum	Maximum	Mean ± SD	95% CI of Mean
Vitamin D (ng/mL)	9.35	47.86	39.35 ± 7.50	37.20–41.50
T3 (ng/mL)	3.04	4.89	3.92 ± 0.66	3.73–4.11
T4 (µg/dL)	0.76	2.88	1.82 ± 0.59	1.65–1.99
TSH (mU/L)	0.01	0.34	0.23 ± 0.11	0.20–0.26

Table 3: Biochemical Parameters in Hyperthyroid Subjects (n = 50)

Comparative Analysis

Comparisons between groups are summarized in Tables 4 and 5.

3.2. Control vs. Hypothyroid (Table 4)

A highly significant reduction in serum vitamin D levels was observed in hypothyroid patients compared to controls (mean difference: 16.37 ng/mL; 95% CI: 13.10–19.64; p < 0.001), indicating a strong association between hypothyroidism and hypovitaminosis D. TSH levels were significantly higher in the hypothyroid group (mean difference: −6.86 mU/L; 95% CI: −8.45 to −5.27; p < 0.001), confirming thyroid hypofunction. T3 levels showed a small but statistically significant difference (p = 0.041), whereas T4 levels did not differ significantly (p = 0.072), suggesting limited alteration in circulating T4 despite elevated TSH.

Parameter	Control (Mean ± SD)	Hypothyroid (Mean ± SD)	Mean Difference (95% CI)	p-value
Vitamin D (ng/mL)	32.64 ± 2.26	16.27 ± 10.50	16.37 (13.10–19.64)	<0.001
T3 (ng/mL)	2.85 ± 0.24	3.05 ± 0.68	-0.20 (-0.41–0.01)	0.041
T4 (µg/dL)	1.28 ± 0.37	1.05 ± 0.24	0.23 (-0.01–0.47)	0.072
TSH (mU/L)	3.17 ± 1.40	10.03 ± 5.61	-6.86 (-8.45– -5.27)	<0.001
Footnote: Independent Student's t-test applied. *p* < 0.05 considered statistically significant.				

Table 4: Comparison Between Control and Hypothyroid Groups

3.3. Control vs. Hyperthyroid (Table 5)

Hyperthyroid patients exhibited significantly elevated T3 and T4 levels compared to controls (both *p* < 0.001), along with markedly suppressed TSH levels (*p* < 0.001), consistent with hyperthyroid physiology. Although mean vitamin D levels were higher in hyperthyroid patients, the difference was not statistically significant (*p* = 0.089), indicating no meaningful association between vitamin D status and hyperthyroidism in this cohort.

Parameter	Control (Mean ± SD)	Hyperthyroid (Mean ± SD)	Mean Difference (95% CI)	p-value
Vitamin D (ng/mL)	32.64 ± 2.26	39.35 ± 7.50	-6.71 (-9.10– -4.32)	0.089
T3 (ng/mL)	2.85 ± 0.24	3.92 ± 0.66	-1.07 (-1.28– -0.86)	<0.001
T4 (µg/dL)	1.28 ± 0.37	1.82 ± 0.59	-0.54 (-0.74– -0.34)	<0.001
TSH (mU/L)	3.17 ± 1.40	0.23 ± 0.11	2.94 (2.53–3.35)	<0.001

Table 5: Comparison Between Control and Hyperthyroid Groups

3.4. Overall Interpretation of Tables

The tabulated findings demonstrate a clear and consistent pattern across study groups. Hypothyroid patients exhibit significantly reduced serum vitamin D levels in conjunction with elevated TSH concentrations, supporting a potential link between hypovitaminosis D and thyroid hypofunction. In contrast, hyperthyroid patients show expected hormonal alterations without evidence of vitamin D deficiency.

The confidence intervals and standard deviations further highlight variability within groups, particularly among hypothyroid individuals, suggesting that vitamin D deficiency may vary in severity and could be influenced by additional factors such as environmental exposure or disease duration.

Summary of Key Findings

- Vitamin D levels were significantly lower in hypothyroid patients compared to controls.
- TSH levels were significantly elevated in hypothyroidism and suppressed in hyperthyroidism.
- T3 and T4 levels were significantly elevated in hyperthyroid patients.
- No statistically significant association was observed between vitamin D levels and hyperthyroidism.

4. Discussion

Vitamin D deficiency is a well-recognized global health concern, affecting approximately one billion individuals worldwide. According to the Endocrine Society guidelines, vitamin D deficiency is defined as serum 25-hydroxyvitamin D [25(OH)D] levels <20 ng/mL, while levels of 21–29 ng/mL are considered insufficient [24]. In the present study, hypothyroid patients exhibited significantly lower mean serum vitamin D levels compared with controls, whereas hyperthyroid patients demonstrated values within the normal range. These findings suggest a potential association between hypovitaminosis D and hypothyroidism.

The observed reduction in vitamin D levels among hypothyroid patients is consistent with previous studies. Yasmeh and Mackawy et al. similarly reported significantly lower serum 25(OH)D levels in patients with hypothyroidism, supporting the hypothesis that vitamin D deficiency may be linked to thyroid dysfunction [25,26]. The lack of a significant difference in vitamin D levels between hyperthyroid and control groups in the present study further indicates that this association may be more specific to hypothyroid states.

Vitamin D exerts its biological effects through the vitamin D receptor (VDR), a member of the nuclear steroid hormone receptor family, which is widely expressed in immune cells including macrophages, dendritic cells, and T and B lymphocytes [27]. Through VDR-mediated signaling, vitamin D modulates both innate and adaptive immune responses by suppressing pro-inflammatory T helper cell activity and inhibiting B-cell proliferation and immunoglobulin production [28]. These immunomodulatory properties are particularly relevant in the context of autoimmune thyroid diseases, where dysregulation of immune tolerance plays a central role.

Emerging evidence suggests that vitamin D deficiency may contribute to the pathogenesis of autoimmune thyroid disorders such as Hashimoto's thyroiditis and Graves' disease. For instance, Wang et al. reported significantly reduced serum 25(OH)D levels in patients with newly diagnosed Graves' disease [29]. The pathophysiology of Graves' disease involves immune-mediated stimulation of the thyroid gland through antibodies directed against the TSH receptor, resulting in excessive thyroid hormone production [30]. Vitamin D may exert a protective effect by modulating immune responses and maintaining immune homeostasis.

In the present study, TSH levels were significantly elevated in hypothyroid patients and markedly suppressed in hyperthyroid patients, consistent with the established negative feedback regulation of the hypothalamic–pituitary–thyroid axis [31]. These findings reinforce the role of TSH as a sensitive biomarker for thyroid dysfunction. Clinically, TSH remains the primary screening tool, with levels >10 mU/L strongly suggestive of hypothyroidism, while suppressed levels (<0.1 mU/L) warrant further evaluation for hyperthyroidism [32].

Although alterations in T3 and T4 levels were observed across study groups, these changes were less pronounced compared to TSH, particularly in hypothyroid patients. This observation supports current clinical practice favoring the measurement of free thyroid hormones (fT3 and fT4), which more accurately reflect biologically active hormone fractions compared to total hormone levels [33].

The findings of this study should be interpreted in light of certain limitations. Vitamin D status can be influenced by multiple confounding factors, including dietary intake, sunlight exposure, geographic location, and comorbid conditions. Additionally, the cross-sectional design limits the ability to establish causality between vitamin D deficiency and thyroid dysfunction. Despite these limitations, the study provides important evidence supporting an association between hypovitaminosis D and hypothyroidism.

5. Conclusion

The present study demonstrates a significant association between reduced serum vitamin D levels and hypothyroidism, suggesting a potential role of vitamin D in the pathophysiology of thyroid disorders, possibly through immunomodulatory mechanisms. In contrast, no significant association was observed between vitamin D status and hyperthyroidism.

Given the high prevalence of vitamin D deficiency, assessment of vitamin D status in patients with hypothyroidism may have clinical relevance. Vitamin D supplementation may be beneficial in deficient individuals; current recommendations suggest treatment with 50,000 IU of vitamin D_2 or D_3 weekly for eight weeks or an equivalent daily dose to achieve serum levels above 30 ng/mL.

However, the clinical utility of vitamin D supplementation specifically for thyroid disease management remains uncertain. Further large-scale, longitudinal, and interventional studies are required to clarify the causal relationship and to determine whether vitamin D supplementation can play a therapeutic role in the prevention or management of thyroid disorders.

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