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Deficiency of Vitamin D in Patients with Hypothyroidism and as a Related Risk Factor for Cardiovascular Disease

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Abstract: Hypothyroidism is a common health condition caused by a deficiency of the thyroxine hormone. The thyroxine hormone regulates the body's metabolic functions, and its deficiency can result in complications across various organs. Vitamin D deficiency is a worldwide concern that has health implications; it is regarded as a pandemic. It represents a global health concern primarily stemming from insufficient sunlight exposure. Hypothyroidism has been demonstrated to adversely affect the cardiovascular system, leading to issues such as vascular endothelial dysfunction, elevated systemic vascular resistance, dyslipidemia, and early-onset atherosclerosis beyond the typical symptoms. Objectives: To examine the frequency of vitamin D insufficiency in individuals with hypothyroidism and assess its possible function as an independent or contributing risk factor for cardiovascular ailments. A cross-sectional study was conducted at Azadi teaching hospital between February 2026 and October 2026. Methodology: A cohort of 90 participants aged between 25 and 65 was assembled and classified into two groups: hypothyroidism group and control group. Venous blood samples were collected after an overnight fast lasting 8 to 12 hours. The following biomarkers were evaluated: Serum TSH and FT4, Serum 25(OH)D concentrations, Fasting Lipid Panel (Total Cholesterol [TC], Low-Density Lipoprotein [LDL-C], High-Density Lipoprotein [HDL-C], Triglycerides [TG], and High-Sensitivity C-Reactive Protein [hs-CRP]). The data analysis was performed using SPSS Version 26.0. Continuous variables were expressed as Mean $\pm$ Standard Deviation (SD). Results: Serum 25(OH)D concentrations were significantly lower in patients with hypothyroidism than in healthy subjects. The hypothyroidism group showed a significantly higher prevalence of dyslipidemia and increased inflammatory markers. Pearson correlation assessment showed a statistically significant negative link between blood 25(OH)D and TSH ($r = -0.42$, $p < 0.001$, and between 25(OH)D and LDL-C ($r = -0.38$, $p = 0.002$). Conclusion: Vitamin D insufficiency was shown to be quite common in primary hypothyroidism patients and was substantially linked with more severe dyslipidemia and elevated inflammatory markers (hs-CRP). These accumulated factors greatly increase the risk for subsequent cardiovascular diseases.

Keywords: Vitamin D, Hypothyroidism, Cardiovascular Disease

1. Introduction

Hypothyroidism is a prevalent medical disorder resulting from an insufficiency of the thyroxine hormone. The thyroxine hormone governs the body's metabolic processes, and its insufficiency may lead to difficulties in multiple organs. Untreated, it can result in grave health consequences and ultimately culminate in mortality. Numerous instances of hypothyroidism exist within the general populace, and these instances exhibit a deficiency in symptom specificity. Consequently, hypothyroidism is primarily characterised as a

metabolic condition [1]. Primary hypothyroidism is characterised by elevated thyroid-stimulating hormone (TSH) levels in the bloodstream, above the normal range, alongside diminished concentrations of free thyroxine (T4) in the blood, falling below the normal range. Subclinical hypothyroidism represents a modest kind of hypothyroidism. Individuals with subclinical hypothyroidism exhibit TSH levels exceeding the normal range (5-10 μ IU/L) while maintaining free T4 levels within the standard reference range [2].

Vitamin D is a lipid-soluble vitamin that is notably significant in potentially reducing the risk of several chronic ailments, such as autoimmune disorders, infectious diseases, endocrine issues, and cardiovascular abnormalities [3]. Vitamin D insufficiency is a global issue that carries health ramifications; it is considered a pandemic [4]. It constitutes a worldwide health issue mostly resulting from inadequate sunshine exposure [5]. The primary sources of vitamin D include sunlight, dietary intake, and supplementation, which are deemed physiologically inert and require activation via two successive enzymatic hydroxylation processes in the liver and kidneys [6]. The vitamin D receptor (VDR) is present in several bodily tissues, such as the brain, heart, skin, gastrointestinal system, gonads, prostate, breast, immune cells, bone, kidney, and thyroid gland. In hypothyroidism, the thyroid gland does not make enough thyroid hormones. This results in a generalised slowing down of metabolic activities [7].

In addition, hypothyroidism has been shown to have deleterious consequences on the cardiovascular system such as vascular endothelial dysfunction, increased systemic vascular resistance, dyslipidemia and premature atherosclerosis outside the usual symptoms [8].

At the same time, Vitamin D (25-hydroxyvitamin D) has evolved from its conventional role in calcium-phosphate homeostasis to a potent pleiotropic hormone [9]. Vitamin D receptors (VDR) are present in many organs, including vascular endothelial cells, cardiomyocytes and immunological cells. Emerging evidence supports a twofold relationship [10].

1. Thyroid-Vitamin D Axis

Vitamin D insufficiency is common in autoimmune and non-autoimmune thyroid illnesses and may modulate thyroid autoimmunity and TSH expression.

2. Cardiovascular Risk

Low levels of Vitamin D have been linked to hypertension, heart hypertrophy, vascular calcification and systemic low grade inflammation.

Although these individual associations are known, the joint association of Vitamin D insufficiency and primary hypothyroidism in the contribution to cardiovascular risk has not yet been fully evaluated in the clinical setting. Objective: The present study was aimed to investigate the status of Vitamin D in hypothyroid patients and to find out if Vitamin D insufficiency in hypothyroid patients aggravates the markers of cardiovascular disease risk.

2. Materials and Methods

2.1. Study Population and Design

A cross-sectional analysis was performed at Azadi teaching hospital from February 2026 to October 2026. A total of 90 individuals aged 25 to 65 were recruited and divided into two categories:

Group A (Hypothyroid group): 60 individuals with confirmed primary hypothyroidism (elevated TSH, decreased/normal FT4).

Group B (Control Group): 30 healthy persons matched for age and gender to patients.

Exclusion Criteria: Individuals with a history of coronary artery disease, chronic renal impairment, liver dysfunction, primary hyperparathyroidism, or those who have utilised Vitamin D/calcium supplements or lipid-lowering medications in the preceding 6 months were omitted.

2.2. Biochemical Examination

Venous blood specimens were obtained following an overnight fast of 8 to 12 hours. The subsequent biomarkers were assessed:

Thyroid Assessment: Serum TSH and FT4 evaluated.

Vitamin D Status: Serum 25(OH)D levels assessed with mini vidas. Categories were designated as: Deficiency: < 20 ng/mL}

Insufficiency: 20–29 ng/mL}

Sufficiency: ≥ 30 ng/mL}

Cardiovascular & Metabolic Markers Fasting Lipid Panel (Total Cholesterol [TC], Low-Density Lipoprotein [LDL-C], High-Density Lipoprotein [HDL-C], Triglycerides [TG] and High-Sensitivity C-Reactive Protein (hs-CRP).

2.3. Statistical Analysis

The analysis of data was conducted with SPSS Version 26.0. Continuous variables were represented as Mean ± Standard Deviation (SD). The Student's t-test and ANOVA were utilised for comparative analysis among groups. Pearson's correlation coefficient (r) was employed to assess relationships among variables. A p-value less than 0.05 was deemed statistically significant.

3. Results

3.1. Fundamental Biochemical Metrics

Patients with hypothyroidism had markedly reduced serum 25(OH)D concentrations in comparison to healthy individuals. Dyslipidemia and heightened inflammatory markers were markedly more prevalent in the hypothyroidism cohort, see Table 1.

Table 1. Comparison of Thyroid Function, Vitamin D Levels, Lipid Profile, and Inflammatory Markers Between the Hypothyroidism and Control Groups

parameters	Hypothyroidism group (n=60)	control group (n=30)	p value
TSH(μIU/ml)	10.1 ± 3.6	2.6 ± 1.1	<0.001
25(OH)D (ng/ml)	17.8 ± 4.6	30.8 ± 6.1	<0.001
Total cholesterol(mg/dl)	233.5 ± 39.2	179.4 ± 27.1	<0.001
LDL-C (mg/dl)	143 ± 25.6	99.3 ± 16.5	<0.001
Triglyceride (mg/dl)	178.2 ± 39.5	124.6 ± 25.1	<0.001
hs-CRP (mg/L)	3.45 ± 1.12	1.15 ± 0.42	<0.001

3.2. Influence of Vitamin D Concentrations on Cardiovascular Indicators in Hypothyroidism

Subgroup examination within the hypothyroid population indicated that those with pronounced Vitamin D insufficiency (< 20 ng/mL) demonstrated much worse lipid profiles and heightened systemic inflammatory indicators relative to hypothyroid patients with adequate Vitamin D (≥30 ng/mL).

Principal Discovery: Pearson correlation assessment revealed a statistically significant negative connection between blood 25(OH)D and TSH ($r = -0.42$, $p < 0.001$), in addition to a similar association between 25(OH)D and LDL-C ($r = -0.38$, $p = 0.002$).

4. Discussion

The results of this research emphasize a notable correlation between primary hypothyroidism and Vitamin D insufficiency, accentuating their combined influence on elevating the risk of secondary cardiovascular diseases.

1. Pathophysiological association of Vitamin D with Hypothyroidism:

Vitamin D exerts an influence on immune modulation. Insufficiency may hinder immunological tolerance, intensifying the death of thyroid tissue in autoimmune disorders (Hashimoto's thyroiditis). Moreover, insufficient Vitamin D may disrupt the regulation of thyroid hormone receptors and hinder intestinal calcium absorption, both of which are indirect factors affecting thyroid gland metabolism [11], [12]. Mackawy et al. conducted a comprehensive cross-sectional analysis of patients with hypothyroidism, revealing a strong association between hypovitaminosis D and increased serum TSH levels when compared to euthyroid controls ($p < 0.001$), corroborating an inverse relationship consistent with our results [13], [14].

2. Synergistic Impact on Cardiovascular Risk:

Atherogenic Dyslipidemia: Hypothyroidism impedes the clearance of LDL-C due to diminished expression of LDL receptors. A deficiency in vitamin D intensifies this state by modifying lipid metabolism via the modulation of lipolysis and the hormonal activity of adipose tissue [13]. Chaudhuri et al indicated a negative link between serum 25(OH)D and lipid metrics, revealing an inverse relationship with LDL, which aligns with your observation.

Endothelial Dysfunction and Inflammation: Increased hs-CRP levels in hypothyroid individuals with Vitamin D insufficiency indicate systemic low-grade inflammation. Vitamin D typically suppresses nuclear factor-kappa B (NF- κ B) pathways; its lack permits unregulated inflammatory signalling, leading to endothelial damage and the initiation of atherosclerotic plaque formation [15].

5. Conclusion

Vitamin D insufficiency is notably common among individuals with primary hypothyroidism and is significantly associated with worsened dyslipidemia and increased inflammatory indicators (hs-CRP). These cumulative elements markedly enhance the risk assessment for secondary cardiovascular ailments.

Recommendation

Regular assessment for Vitamin D insufficiency ought to be contemplated in the therapeutic approach to hypothyroidism. Adjuvant Vitamin D supplementation could serve as a significant therapeutic approach to enhance metabolic equilibrium and reduce cardiovascular disease risk in this demographic.

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