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Evaluation Of Vitamin D3 And Interleukin 6 In Children With Thyroid Disorders In Thi-Qar Province/ Iraq

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ABSTRACT:
Objective: *The study aims to determine the relationship between thyroid disorders and the concentration of vitamin D3, Interleukin-6, TSH, T3, T4 hormones, Liver enzyme (AST, ALP, and ALT) and kidney functions (Uric acid, Creatinine, and Urea) in blood samples of children in Thi-Qar Province, Iraq.*
Methods: *The study sample was divided into three groups: the first group (control), the second group (hypothyroidism) and the third group (hyperthyroidism). The above-mentioned criteria were measured for the three groups according to the work methods specified in the detection tools.The study sample consisted of the patients attending Al-Rifai Teaching Hospital and Endocrinology center in Thi-Qar, from August 4, 2024, to June 10, 2025.*
Results: *The results of this study showed a significant increase in Vit. D3, TSH and IL-6 in the second group. Also, there is a significant increase in Vit. D3, T4, ALT, urea, and uric acid in the third group when compare with the first group and there is a significant decrease in AST and ALP in the second and third groups compare with first group, while a significant decrease in T4 in the second group.*
Conclusion: *There is a clear and reciprocal effect between thyroid disorders and vit-D3, IL-6, TSH, T3, T4, Urea, Uric acid, ALT, AST, ALP.*
Keywords*: Thyroid disorders, Hyperthyroidism, Hypothyroidism, Interleukin-6, Vitamin D3*

1.INTRODUCTION:

Thyroid diseases are considered the second most common metabolic disorders worldwide after diabetes. Various factors, including geographical and biological variations, affect the prevalence rates in different parts of the world[1]. Hormonal disorders are classified according to their levels into hypothyroidism at low hormonal levels and hyperthyroidism at high levels. [2]. Hypothyroidism occurs due to an abnormal decrease in thyroid hormone concentrations, either because of the gland itself or due to insufficient stimulation from the hypothalamic-pituitary axis. It is one of the most common and widespread thyroid disorders worldwide and increases proportionally with the growing global population [3]. Hypothyroidism, which has a prevalence rate ranging from 0.3-3.7% in the general population in the United States and from 0.2-5.3% in Europe, may manifest with symptoms such as unexplained weight gain, fatigue, cold intolerance, delayed growth, or puberty. Additionally, the development of hypothyroidism is influenced by multiple factors such as genetic variations, medications, infections, environmental factors, tumors or diseases or some of their treatments, and nutrition [4]. Hyperthyroidism refers to excessive concentrations of thyroid hormones in the tissues due to increased biosynthesis and secretion of thyroid hormones [5]. The most common causes of hyperthyroidism are toxic adenoma, toxic multinodular goiter, painless (silent) thyroiditis, and Graves' disease (GD), which is the most common cause in the United States [6]. Hyperthyroidism accounts for about 15% of thyroid disorders in children, with most cases attributed to autoimmune hyperthyroidism [7]. The onset of the disease often occurs between the ages of 20 and 50, and it is more common in women in the case of GD, while in patients with toxic adenoma, the disease is more common in individuals over 50 years old. Cytokine levels increase in both autoimmune and non-autoimmune hyperthyroidism, such as (Interleukin-18 (IL-18), Tumor necrosis factor alpha (TNF-α), Interleukin-6 (IL-6)), indicating that this may result from the chronic effects of excessive thyroid hormone rather than the accompanying autoimmune inflammatory condition present in GD. There are very few studies that emphasize the importance of serum cytokines in hyperthyroidism, which have taken into account the role of age, which seems to be a critical variable, significantly and directly associated with many cytokines. Therefore, it should be considered that patients with toxic nodular goiter (TNG) are usually older than patients with GD [8]. One of the important nutrients is vitamin D3, which performs various functions through receptors expressed in many body organs. There are several mechanisms through which vitamin D3 can modulate the concentrations of

thyroid-stimulating hormone (TSH) and thyroid hormones. Experimental research indicates that vitamin D3 directly affects type 2 iodothyronine deiodinase (Dio2), an enzyme required to convert thyroxine (T4) to Triiodothyronine (T3) in target tissues [9].

Aim of study: The study was designed to research the relationship between thyroid disorders and the concentration of Vit. D3, Interleukin-6, TSH, T3, T4, Liver enzyme (AST, ALP, and ALT), and kidney functions (Uric acid, Creatinine, and Urea) in children blood with thyroid disorders.

2.METHODS:

2.1. Collecting blood samples and methods

The study sample included 30 females aged between 1 to 15 years who were attending Al-Rifai General Hospital and Endocrinology center in Thi-Qar. The blood samples were collected periodically through direct collection from the patient and then left at room temperature for 7 minutes to allow the blood to clot. Thereafter, the samples were placed in a centrifuge and set to 3500 RPM for 5 minutes. The resulting serum was collected in microtubes and stored in the freezer (-20 degrees Celsius) for subsequent chemical tests.

2.2. Measuring TSH, T3 and T4

All these hormones measured by using Maglumi800 device from Snaibe company.

2.3. Measuring liver enzyme and kidney functions

Liver enzyme (AST, ALT, and ALP) and kidney functions (Urea, Uric acid, and Creatinine) were measured by using Semi-auto biochemical analyzer new 21A device from Genex company.

2.4. Measuring Vitamin D3

Vitamin D3 was measured by ichroma device from Boditech Med company.

2.5. Measuring Interlukin-6

Interlukin-6 was measured by ELISA device from Biotek company.

2.6. Statistical analysis

Statistical analysis was performed by SPSS version 27. The results were expressed as mean ± standard deviations (mean ± SD). One-way ANOVA was used to compare parameters in different studied groups. P-value ($P \leq 0.05$) was considered statistically significant.

3.RESULTS AND DISCUSSION

3.1. Functions of thyroid gland

The results of the study in **Table (1),** showed a significant increase in the concentration of T4 in the third group and a significant decrease in the second group compared with control group. As for the concentration of T3, there was non- significant increase in third group and non-significant decrease in second group compared with control group. The results also indicated a significant increase in the concentration of TSH in the second group and a significant decrease in the third group compared with control group ($P \leq 0.05$).

Table (1): The concentrations of T4, T3, and TSH (mean± SD)

Groups / Parameters	Group 1 (Control group)	Group 2 (Hypothyroidism)	Group 3 (Hyperthyroidism)
Thyroxine (T4) (µg/dL)	8.183 b ±0.893	6.575 c ±1.424	11.894 a ±0.785
Triiodothyronine (T3) (nmol/L)	1.200 a ±0.216	1.187 a ±0.752	1.727 a ±0.715
Thyroid-stimulating hormone (TSH) (uIU /ml)	1.555 b ±0.453	7.250 a ±1.323	0.421 c ±0.471

The different letters refer to a significant difference at (P≤ 0.05).

T4 and T3 can have a negative feedback effect on TSH levels. Pirahanchi et al. (2018) indicated that high T3/T4 levels reduce TSH secretion from the anterior pituitary, while conversely, low T3/T4 levels increase TSH secretion. T3 is the primary inhibitor of TSH secretion, and because TSH levels are very sensitive to small changes in free T4 through a negative feedback loop, abnormal TSH levels are detected earlier than free T4 levels in both hypothyroidism and hyperthyroidism. There is a logarithmic linear relationship between T3/T4 and TSH, and small changes in T3/T4 lead to large changes in TSH [10] According to a study by Ortiga-Carvalho et al., 2011, the thyroid hormones T4 and T3 control the secretion of TRH and TSH through negative feedback to maintain physiological levels of the master hormones of the hypothalamic pituitary thyroid axis. Low circulating TH levels due to primary thyroid failure lead to increased TRH and TSH production, while the opposite occurs when circulating TH levels are increased. Several neural, humoral, and local factors modulate the HPT axis, and in certain cases, alterations in the physiological function of the axis can determine the vital role of TH hormones in nervous system development, linear growth, energy metabolism, and thermogenesis. TH hormones also regulate hepatocellular nutrient metabolism, fluid balance, and the cardiovascular system. T3 is the preferred ligand for THR, while T4, which has a serum concentration 100 times higher than T3, undergoes extra thyroidal conversion to T3, catalyzed by 5'-deiodinase D1 and D2, enzymes that activate thyroid hormones [11]. These results are consistent with the results of the study by Mortoglou & Candiloros, 2004, that showed a significant increase in T4 hormone levels in patients with hyperthyroidism and a significant decrease in patients with hypothyroidism compared to the control group. As for TSH, it was significantly elevated in patients with hypothyroidism, unlike its low levels in patients with hyperthyroidism [12]. In addition, our results are not agree with the results of a comparative study by Choudhury, 2016, that showed T4 and T3 levels in patients with nephrotic syndrome and hypothyroidism were significantly lower, while TSH levels were elevated in the same group [13]. However, our results are consistent with the results of a cross-sectional study by Ayala-Moreno et al., 2018, this study showed significant differences between participants, where T4 was elevated in hyperthyroid patients and TSH was elevated in hypothyroid patients [14]. According to the study conducted by Khaleghzadeh-Ahangar et al., 2022, any disorder in thyroid secretions, whether it be a deficiency or an increase, can occur as a result of a defect in the secretion of TSH from the pituitary gland. This defect may be an increase in the secretion of TSH, which positively affects the secretion of T4 to the point that the person may suffer from hyperthyroidism. Sometimes the defect may be in the thyroid gland itself, which causes an increase or decrease in the preparation of thyroid hormones. Increased inappropriate stimulation may cause a tumor in the pituitary gland, which causes an increase in the secretion of TSH, which is unresponsive to negative balanced nutrition. It may also result from the pituitary gland's resistance to thyroid hormone or autoimmune diseases. The defect may also be in the preparation of iodine, resulting either from outside the body through a deficiency or quality in food or from a defect in its absorption by the thyroid gland. A deficiency in it will lead to the loss of the basic factor for the production of thyroxine [15].

3.2. The relationship between thyroid disorders and liver enzymes

The results in **Table (2)**, showed a significant decrease in the concentration of aspartate transaminase (AST) and alkaline phosphatase (ALP) in the second and third groups compared with control group, and a significant increase in the concentration of alanine transaminase (ALT) in third groups compared with second group and control group at a probability ≤0.05

Table (2): The relationship between thyroid disorders and liver enzymes (mean ± SD)

Groups / Parameters	Group 1 (Control group)	Group 2 (Hypothyroidism)	Group 3 (Hyperthyroidism)
Aspartate transaminase AST (U/L)	24.500 a ±5.089	17.875 b ±1.726	18.111 b ±2.472
Alkaline phosphatase ALP (U/L)	150.500 a ±40.213	92.875 b ±5.962	100.666 b ±1.500
Alanine transaminase ALT (U/L)	22.000 b	20.125 b	31.222 a

	±4.690	±1.807	±4.236

The different letters refer to a significant difference at (P≤ 0.05).

According to a study by Hasan et al., 2024, Thyroid hormones regulate the metabolism of all cells, including liver cells, and thus will affect liver function. The liver and thyroid are two of the most important organs due to their physiologically complex interconnected relationships involving many biochemical processes. Therefore, any problem in one negatively affects the other. When thyroid dysfunction is treated, thyroid hormones return to normal and the body's metabolism changes. This temporary change can affect liver function and potentially affect the liver. As a result, hyperthyroidism can alter the amount of liver enzymes and damage liver cells. It makes sense that diseases of these two organs interact or affect each other. Numerous clinical and laboratory studies have linked thyroid and liver problems [16]. A histomorphological study by Nechyporuk et al., 2020, revealed the degeneration of liver cells in hypothyroidism and hyperthyroidism. It showed that the cells destroyed by thyroid disorders were more evident in hypothyroidism than in hyperthyroidism. Both conditions cause changes in the liver structure upon histological examination. This observation may indicate that the degeneration seen in liver cells was not significant enough in hyperthyroidism to modify it, while cell damage is prominent in hypothyroidism. It was noted that hypothyroidism plays a greater role in liver dysfunction [17]. Liver damage can occur as a result of subclinical physiological effects on liver function, direct toxic effects, or systemic consequences of hyperthyroidism. Some people with chronic liver disease may also develop thyroiditis, hyperthyroidism, or hypothyroidism due to autoimmune factors. Thyroid or other hormonal imbalances may cause changes in thyroid hormone metabolism or tests due to liver disease and related conditions [18]. Scappaticcio et al., 2021, indicated that hypothyroidism is associated with slightly decreased levels of some liver enzymes, including AST and ALP. However, the most common effect of thyroid disorders on liver enzymes is associated with slightly elevated levels of some enzymes in cases of hyperthyroidism [19]. According to Yadav et al., 2013, hyperthyroidism may cause elevated ALP levels in some cases as a result of increased metabolic activity and the effect of thyroid hormones on bones, which leads to increased osteoclast activity and the release of ALP from the bones. As for AST levels, they may be normal or slightly affected, and may be affected based on the overall condition of the liver and muscles [20]. These results do not agree with the results of the comparative study conducted by Barzegar et al., 2024, that indicated the levels of AST and ALT in the second and third groups were significantly higher than in the control group [21]. In the same vein, there is no agreement with the results of the retrospective study by Artemniak-Wojtowicz et al., 2019, where this study observed an increase in the baseline activity of the ALT and AST enzymes in 44% and 32.2% of the children, respectively [22]. However, our study's findings regarding ALP levels are inconsistent with the results of a cross-sectional correlational study by Lee et al., 2021, ALP levels in this study were significantly elevated compared to the results of our study [23]. The results of our study also do not agree with the retrospective study that included the medical records of 354 patients between 2009 and 2019, as the results of this study showed a significant increase in the levels of ALT and AST compared to our results [24]. The difference may be due to the fact that this study focused on the records samples of patient unlike our study.

3.3. The relationship between thyroid disorders and kidney functions

The results in **Table (3),** indicated a significant increase in the concentrations of uric acid and urea in the third group compared with control group and the second group, with no significant difference in creatinine concentration among the three groups at P ≤ 0.05.

Table (3): The relationship between thyroid disorder and kidney functions (mean ± SD)

Groups / Parameters	Group 1 (Control group)	Group 2 (Hypothyroidism)	Group 3 (Hyperthyroidism)
uric acid (mg/dl)	6.083 b ±0.416	6.150 b ±0.616	6.877 a ±0.777
Creatinine (mg/dL)	0 .410 a ±0.220	0 .573 a ±0.242	0.496 a ±0.091

Urea (mg/dL)	26.500 b ±5.282	29.250 ab ±4.166	31.000 a ±2.738

The different letters refer to a significant difference at (P≤ 0.05).

Thyroid hormones are essential for adequate kidney growth and development. Thyroid dysfunction causes marked changes in glomerular and tubular function, as well as in electrolyte and water balance. Hypothyroidism is accompanied by decreased glomerular filtration rate, hyponatremia, and altered water excretion capacity. Excessive thyroid hormone levels lead to increased glomerular filtration rate and renal plasma flow [25]. Kidney disease, in turn, leads to significant changes in thyroid function. Various types of glomerulopathies have been reported to be associated with both hyperthyroidism and hypothyroidism. Nephrotic syndrome is associated with changes in thyroid-stimulating hormone (TH) concentrations due to protein loss in the urine. Acute kidney injury and chronic kidney disease have marked effects on the hypothalamic-pituitary-thyroid axis. Uremia impairs pituitary thyrotropin-releasing hormone (TSH) secretion. In contrast to other non-thyroid chronic diseases, recent research data suggest that thyroid hormone, particularly T3, may be a predictor of survival in kidney disease patients [26]. Also, Helmy, 2020, Pointed out that high uric acid may be caused by increased purine metabolism in the body, as uric acid is produced as a result of the breakdown of purines in the body, which results in increased accumulation of uric acid. Thyroid diseases affect purine metabolism in the body, which may increase the level of uric acid in the serum [27]. Iglesias et al., 2017, indicated that hyperthyroidism is characterized by increased cardiac output, renal plasma flow, and glomerular filtration rate (GFR), leading to decreased serum creatinine levels due to reduced creatinine synthesis and increased renal excretion [28]. The results of our study are inconsistent with those of a cross-sectional study conducted by Mohamed & Sayed, 2023, the study results showed a significant positive correlation between the concentration of TSH and creatinine, and a negative correlation between the concentration of T3 and creatinine [29]. Similarly, the current findings are not consistent with the results of a comparative study by Sayari et al., 2018, That showed higher levels of creatinine (P = 0.003), while serum uric acid levels in children with subclinical hypothyroidism were not significantly different from those in the control group (P = 0.200). According to the results of this study, there was no significant correlation between TSH, T3, and T4 with any of the kidney function parameters [30]. In the same context, it was found that our study does not agree with the results of Garrido-Magaña et al., 2009, that indicated that the incidence rate of thyroid dysfunction in kidney patients is higher than the rate found in the general population without kidney diseases. The results obtained in this study also confirm that pediatric patients with chronic hypothyroidism show a higher incidence rate of kidney diseases (28%) [31]. In contrast, our study found that it aligns with the results of the cross-sectional observational study at the Sultan Hospital in Muscat, Oman, where data were gradually collected for all newly diagnosed chronic kidney disease patients without a known history of thyroid disease from January 2018 to December 2019. This study showed a significant increase in urea levels in the hyperthyroid group. However, creatinine levels did not show any statistical difference [32].

3.4. The relationship between thyroid disorders and the concentrations of vitamin D3 and interleukin 6

The results in **Table (4),** showed a significant increase in the concentration of Vitamin D3 in the second and third groups compared with control group. The results also indicated a significant increase in the concentration of interleukin-6 in the second group compared with third and control groups at a p≤ 0.05.

Table (4) The relationship between thyroid disorder and the concentration of vitamin D3 and interleukin 6 (mean ± SD)

Groups / Parameters	Group 1 (Control group)	Group 2 (hypothyroidism)	Group 3 (hyperthyroidism)
Vitamin D3 (ng/Ml)	6.311 c ±1.370	11.345 b ±2.758	14.688 a ±3.205

interleukin-6 (IL-6) (pg/mL)	8.433 b ±0.960	10.275 a ±1.263	9.777 ab ±1.389

The different letters refer to a significant difference at (P≤ 0.05).

Vitamin D3 plays an important role in the body in terms of regulating calcium and phosphate, which are essential for the body's health. Vitamin D3 deficiency is linked to thyroid disorders. In most cases, there is no direct relationship between hypothyroidism and high vitamin D. However, vitamin D deficiency can lead to increased parathyroid hormone secretion, which can affect vitamin D absorption. Other factors, such as sun exposure or taking vitamin D supplements, can also lead to elevated blood levels. Consulting a doctor is essential to determine the cause of high vitamin D levels in hypothyroidism [33]. The current results regarding vitamin D3 concentration are not consistent with the results of a study conducted on 90 children with endocrine disorders associated with hormone replacement therapy. This study found that serum vitamin D3 concentrations were significantly lower in the second and third groups compared with control group [34]. Similarly, our current findings are inconsistent with a study conducted in Iraq by Obaid et al., 2020. This study showed no significant association between vitamin D3 concentrations in the second and third groups compared with control group [35]. In addition, our results are inconsistent with those of a cross-sectional study that linked children's data records related to routine health checkups to promote early childhood development. This study found a significant association between vitamin D3 deficiency and the risk of hypothyroidism [36] .

Recent studies indicate that monocyte-derived mixed factors such as tumor necrosis factor-A (TNF-a), interleukin-18 (IL-1B), and interleukin-6 (IL-6) modulate the function of the pituitary-thyroid axis in vitro or in vivo and have been associated with intracellular changes in the thyroid function tests that seen in the thyroid disease syndrome. In addition, these cytokines play a key role in inducing several inflammatory responses seen in several no thyroidal diseases, but their clinical significance on thyroid function has not been elucidated. It has been demonstrated that human thyroid cells are capable of synthesizing cytokines that activate beta and beta lymphocytes. These immune cells play an important role in the initiation and persistence of thyroid autoimmunity [37]. As for the concentration of interleukin-6, the current results are not consistent with the results of a study conducted on a group of children with hypothyroidism, as the study showed no significant correlation between the concentration of interleukin-6 and hypothyroidism [37]. Conversely, our results were consistent with those of a cross-sectional study conducted by Nagila et al., 2024. This study found that interleukin-6 were elevated in 20.48% of hypothyroidism cases, demonstrating a significant association between inflammatory status and hypothyroidism at a P<0.01 [38].

6. CONCLUSION:

There is a clear and reciprocal effect between thyroid disorders and vit-D3, IL-6, TSH, T3, T4, Urea, Uric acid, ALT, AST, ALP.

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