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Research Article

Detection of IL-17 in clinical and subclinical hypothyroid Iraqi patients

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Abstract:

The current realization was implemented out to assess the level of Interleukin17 (IL-17) in hypothyroidism Iraqi patients taking into account various thyroid functioning states (Clinical hypothyroidism and subclinical hypothyroidism). Include 84 individuals who have been clinically identified to check hypothyroid disoorder. Three research groups have been examined. Of various ages, there were 48 clinical hypothyroid patients, 13 subclinical hypothyroid patients, and 23 healthy persons. According to this study, of all patients, the percentage of females (83.3%) with subclinical hypothyroidism and the percentage of males (16.7%) with clinical hypothyroidism and subclinical hypothyroidism, respectively, are higher than The T4 level in the subclinical group compared to the level of healthy group is not noteworthy different from the T3 level in any of the three groups, according to the results. When compared to the other two groups' levels, the clinical hypothyroid patients exhibit the lowest level of T4, with a significant difference. When comparing the TSH level of clinical hypothyroid individuals to that of the other two groups, there is a notable difference in the former group.We can notice a significant elevated in level of IL-17 in clinical hypothyroidism, on the contrary, subclinical hypothyroid have a lower level of IL-17 than in healthy subjects.

Keywords: Hypothyroidism, Subclinical hypothyroidism, TSH, IL-17

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INTRODUCTION

Hypothyroidism is a widespread endocrine disease arising from insufficiency of thyroid hormone or its effects on peripheral tissues. According to the causes, hypothyroidism has three types. The thyroid gland's insufficient production of thyroid hormone is typically the cause of primary hypothyroidism.

The secondary hypothyroidism is resulting from inadequate secretion of TSH from the pituitary gland and tertiary hypothyroidism resulting from inadequate secretion of TRH from the hypothalamus Vaidya, B. and (Pearce, 2008).

The cause of hypothyroidism could be (Congenital) which results from abnormality in the production and actions of hormones or (Aquired) which include: autoimmune thyroiditis, iodide deficient diet and thyroid ablation, or (Pharmacological): iodide, propylthiouracil, methimazole, lithium, thiocyanate etc. McPhee S. And (Bauer, 1997).

Hypothyroidism is an extremely prevalent illness that affects 3-5% of people (Price A and Weetman 2001).

Hypothyroidism is one of the most endocrine disorder which may result minor symptoms like sadness, cold sensitivity, obese and fatigue, additionally more intense conditions like myxedema and mortality (Chaker et.al, 2017) , (Calissendorff J and Falhammar H 2020).

Subclinical Hypothyroidism (SCH) is defined as a condition in which the level of both hormones (T3, T4) is at their normal level with increased in TSH level above the usual level. (Cooper 2001).

Although there is disagreement on the maximum TSH level, 4 mIU/L is generally accepted as the cutoff; 90% of patients with SCH have modest TSH levels (4.0–10.0 mIU/L) (Duntas, 2019). In patients with SCH, a considerable rise in TSH (i.e., >10.0 mIU/L) has been determined to be an appropriate threshold for starting treatment (Bekkering et al 2019). Yet a 7 mIU/L cut-off is linked to a higher risk of overt hypothyroidism (Li X et al, 2017).

The most common autoimmune condition is called Hashimoto's thyroiditis (HT), sometimes referred to as chronic lymphocytic or autoimmune thyroiditis. Clinical symptoms of HT frequently include an enlarged thyroid, lymphocytic infiltration, and elevated autoimmune antibodies (Bliddal S and Nielsen CH (2017) (Ralli et al, 2020).

Human Interleukin 17 (IL-17), a 15-20 kDa polypeptide with variable glycosylation that is a member of the IL-17 family of cytokines, is the signature cytokine of the recently identified T helper 17 (TH17) cell subset. It is also referred to as IL-17A and cytotoxic T lymphocyte-associated antigen (CTLA-8). Its other name, CTLA-8, came from research done on rodents in which a rat T cell lymphoma cell line and a mouse cytotoxic cell line fused to form an active hybridoma (Gaffen, 2009), (Michalaki et al, 2006).

MATERIAL AND METHODS

Between March 2023 and September 2023, a prospective study was conducted at the Endocrine gland and diabetes center in Baghdad. The study involved 82 participants who had been clinically classified as hypothyroid. Three research groups have been examined: (12 subclinical hypothyroid people and 47 clinical hypothyroid patients, 23 healthy person) of various ages.

Following a 12- to 14-hour fast, venous blood samples (4 ml) were drawn from each participant in the hypothyroid patient and healthy control groups. The samples were centrifuged at 3000 rpm for 10 minutes.

The serum that was collected was utilized in accordance with the manufacturer's protocol to check the biochemical and immunological parameters. Ush0ing kits from Biomerieux (France), the levels of T3, T4, and TSH were evaluated as markers of thyroid function. ELISA kits from R&D (USA) were used to estimate IL-17.

The SPSS (Standard statistical application, version 24) was utilized for all computations and to examine the differences in the research parameters between the three groups. In this investigation, data were represented as mean ± SE, with $P<0.05$ being regarded significant and $P<0.01$ as very significant. One way the results of the analysis of variance were employed, along with using Least Significant Difference (LSD) to significantly compare means substantially.

RESULTS AND DISCUSSION

Table 1 Results showed no noteworthy difference in the level of T3 hormone between the three groups, no noteworthy differences in the level of T4 hormone in the subclinical group compared to the healthy group, and the clinical hypothyroidism patients exhibit least level of Thyroxine (T4) hormone with a noteworthy difference between its level in the other remaining groups and the highest TSH level with a noteworthy difference between its level in the other two groups.

Table 1. The level of thyroid hormones in the blood serum of the three diagnosed groups is described as follows (mean± standard deviation)

Group	Thyroid function test		
	Triiodothyronine T3 (nmol/L)	**Thyroxine T4 (nmol/l)**	**Thyroid Stimulating hormone TSH (mulu/ml)**
Healthy	**1.36 ± 0.084**	**83.09 ± 1.84 b†**	**1.51 ± 1.16 b**
Subclinical hypothyroidism	**1.55 ± 0.195**	**97.55 ± 6.74 ab**	**10.25 ± 3.78 ab**
Clinical hypothyroidism	**1.35 ± 0.075**	**74.54 ± 3.55 c**	**18.25 ± 3.64 a**
P-Value	**0.422**	**0.004****	**0.004****

Data presented as mean ± SE , * significant differences ($p<0.05$),), (**)highly significant differences ($p<0.01$). †Means that do not share a letter (vertically) are significantly different.

The hallmark of hypothyroidism is a higher-than-normal serum level of TSH hormone, which is caused by a decrease in thyroid hormones (T3, T4) in the blood. This condition is explained by the negative feedback process. The development of immunological responses in opposition to the thyroid gland that result in death of glandular cells is one of the most significant factors that might cause hypothyroidism and low levels of Triiodothyronine (T3) and Thyroxine (T4) hormones. Thyroiditis initially manifests as tissue damage that eventually results in gland destruction. Consequently, the gland's capacity to manufacture and distribute thyroid hormones is compromised.

According to our research, there is a markedly higher amount of IL-17 in clinical hypothyroidism. Conversely, compared to healthy persons, subclinical hypothyroidism has a decreased amount of IL-17.

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Pro -inflammatory cytokines (IL-17, IL-17F, IL-22, IL-21) released by a special type of lymphocytes, known (Th17 cells) ,which are essential in long-term inflammatory conditions such systemic lupus erythematosus and asthma. (Traves SL & Donnelly LE, 2008).

“Those cells first described in patients with Gravis disease and found in upper rate in patients are not treated with anti-thyroid drugs” (Nanba et al, 2009). While in Hashimoto disease patients, “it is discovered that the expression of the gene RORC2 responsible for the differentiation subpopulation Th17 phenotype will increased, and improved the number of Th17 cells in peripheral blood and thyroid tissue” (Figueroa-Vega N et al, 2010).

The study of (Idźkowska et al, 2012) found that children with freshly diagnosed Hashimoto thyroiditis had significantly higher levels of IL-17 and IL-23; this finding supports the role of Th17 in the pathophysiology of this illness.

(Wang et al, 2013) found that the blood of individuals with Hashimoto thyroiditis had higher levels of proinflammatory leptin, which may activate the immune system to produce Th17 by stimulating T cell proliferation. In patients with hashimoto thyroiditis, there was a significant correlation found between the expression of IL-17 and thyroid stromal fibrosis, indicating that the proinflammatory effect of IL-17 propels the development of thyroid tissue in the direction of the illness.

(Dapeng et al, 2013) and (Shi et al, 2010) discovered that the peripheral blood cells of Hashimoto's patients had greater levels of IL-17 mRNA expression. This finding led the researchers to propose that Th-17 cells, as opposed to Th1 cells, may be more important in the pathophysiology of Hashimoto's illness.

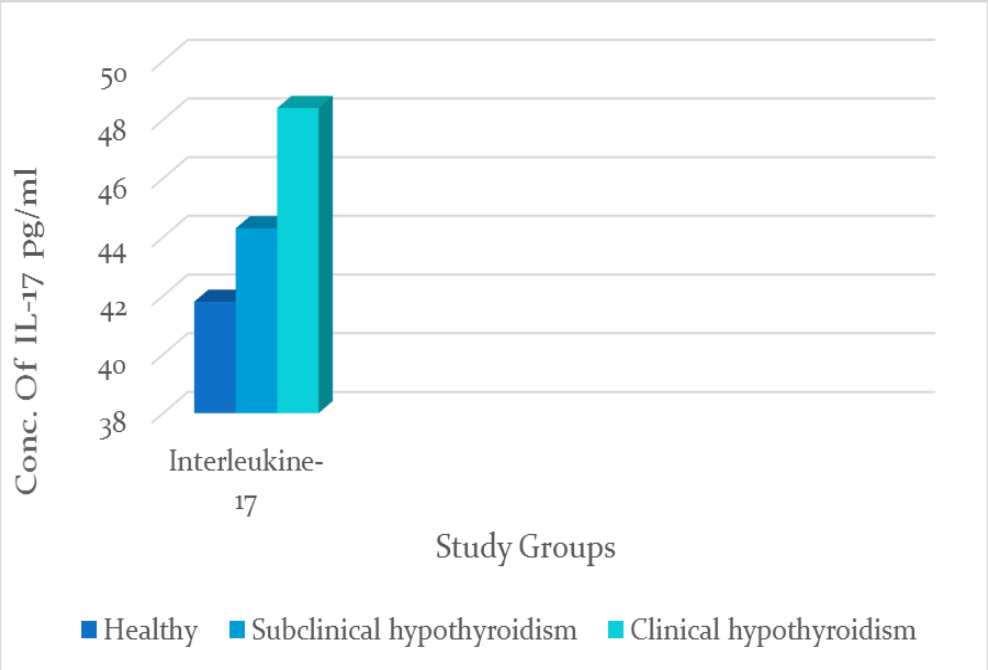

Figure 1. Serum concentration of IL-17 (pg/mL) in the patient's groups incomparison with he control**.**

REFERENCES

Vaidya, B. and Pearce, S.H. (2008), Management of hypothyroidism in adults. BMJ, 337, 284–289.

McPhee S. and Bauer D.C. (1997), Pathology of disease. New York, Lange Medical Books/McGraw-Hill.

Price A and Weetman AP. (2001), Screening for central hypothyroidism is unjustified. BMJ; 322:798. Primary Care. 31(3):321-340.

Chaker. L, Bianco AC, Jonklaas J, Peeters RP. (2017), Hypothyroidism. Lancet; 390 (10101): 1550–1562.

Calissendorff J, Falhammar H. (2020), To treat or not to treat subclinical hypothyroidism, what is the evidence? Medicina (Kaunas); 56(1): 40.

Cooper DS. Clinical practice. Subclinical hypothyroidism. N Engl J Med 2001;345:260–5.

Duntas LH, Yen PM. (2019), Diagnosis and treatment of hypothyroidism in the elderly. Endocrine; 66:63–9.

Bekkering GE, Agoritsas T, Lytvyn L, Heen AF, (2019), Feller M, Moutzouri E, et al. Thyroid hormones treatment for subclinical hypothyroidism: A clinical practice guideline. BM; 365: l2006.

Li X, Zhen D, Zhao M, Liu L, Guan Q, Zhang H, et al. (2017), Natural history of mild subclinical hypothyroidism in a middle-aged and elderly Chinese population: A prospective study. Endocr J; 64:437–47.

Bliddal S, Nielsen CH, (2017), Feldt-Rasmussen U. Recent Advances in Understanding Autoimmune Thyroid Disease: The Tallest Tree in the Forest of Polyautoimmunity. F1000 Res 6: 1776. doi: 10. 12688/f1000research. 11535.1

Ralli M, Angeletti D, Fiore M, D'Aguanno V, Lambiase A, Artico M, et al.. (2020) Hashimoto's Thyroiditis: An Update on Pathogenic Mechanisms, Diagnostic Protocols, Therapeutic Strategies, and Potential Malignant Transformation. Autoimmun Rev 19 (10): 102649. doi: 10.1016/j.autrev.2020 .102649.

Gaffen, S.L. Structure and signaling in the IL-17 receptor family Nat. Rev. Immunol. (2009) , 9 (8):556- 567.

Michalaki, M.A ; Vagenakis, A.G ; Leonardou, A.S ; Argentou, M.N ; Habeos, I.G ; Makri, M.G.; Psyrogiannis, A.L; Kalfarentzos, F.E. and Kgriazopoulou, V. E. (2006) , Thyroid function in humans with morbid obesity. Thyroid.16:73-78.

Zaletel, K. and Gaberscek, S. (2011), Hashimoto's Thyroiditis: From Genes to the Disease. Current Genomics, 12: 576-588

Traves SL, Donnelly LE. (2008), Th17 cells in airway diseases. Curr Mol Med treatment. Thyroid 17: 453-460.

Nanba T, Watanabe M, Inoue N. (2009) Increases of the Th1/Th2 cell ratio.

Figueroa-Vega N, Alfonso-Perez M, Benedicto I (2010), Increased circulating pro-inflammatory cytokines and Th17 lymphocytes in Hashimoto’s thyroiditis. J Clin Endocrinol Metab ; 2010, 95: 953–962.

https://doi.org/10.58613/jbb432

Idźkowska E, Bossowski A, Noiszewska K . Analiza stężenia. (2012), IL-17 i IL-23 w dynamice chorób autoimmunizacyjnych tarczycy u młodocianych. Endokrynologia Pediatryczna; 3: 17–26. improving in U.S. adults? Diabetes Care. 31(1):81–86.

Wang S., S. E. Baidoo, Y. Liu; (2013), "T cell-derived leptin contributes to increased frequency of T helper type 17 cells in female patients with Hashimot's thyroiditis," Clinical and Experimental Immunology; 171(1): 63–68.

Dapeng Li, Wenqian Cai , Runxia Gu, Yi Zhang, Huafeng Zhang,Ke Tang, Pingwei Xu, Foad Katirai , Wei Shi , Longqiang Wang,Tao Huang, Bo Huang. (2013), Th17 cell plays a role in the pathogenesis of Hashimoto's thyroiditis in patients. Clinical Immunology; (149):411–420.

Shi Y, Wang H, Su Z. (2010), Differentiation imbalance of Th1/Th17 in peripheral blood mononuclear cells might contribute to pathogenesis of Hashimoto's thyroiditis," Scandinavian Journal of Immunology; 72 (3): 250–255.