Immunodiagnostic Role of INF-γ in Hashimoto's Thyroiditis: Correlation with Auto antibody and Inflammatory Markers

Noor al-Huda Saber Sadiq[1,*], Dhifaf Zeki Aziz[1]

[1]University of Kufa, Faculty of Science, Department of Pathological Analyses,Al-Najaf, Iraq.

[*]Corresponding author: nooralhadaa.alkhazrajy@student.uokufa.edu.iq

Received 20 May. 2025, Accepted 19 June. 2025, published 30 Dec. 2025.

DOI: 10.52113/2/12.02.2025/56-68

Abstract: Hashimoto's thyroiditis (HT) is an autoimmune disorder in which progressive thyroid failure is induced by Th1-type immunity, and in particular, interferon-gamma (IFN-γ). Immunological diagnosis of HT was made in the present study by comparing 50 patients with HT and 50 sex- and age-matched controls for serum IFN-γ, IL-18, anti-TPO, anti-Tg, hs-CRP, and TSH levels. Biomarkers were quantified using ELISA and Cobas e411 and statistically evaluated for group comparison, correlation, regression, and ROC curve. All biomarkers were significantly higher in HT patients ($p < 0.001$). IFN-γ was strongly correlated with IL-18, anti-TPO, and anti-Tg ($r > 0.7$) and had high diagnostic accuracy (AUC = 0.92; cutoff = 59.96 pg/mL). These observations implicate IFN-γ as a key mediator of HT pathogenesis and suggest its utility in diagnosis and in the design of future immunomodulatory treatment strategies.

Keywords: Hashimoto's thyroiditis, interferon-gamma, interleukin-18, hs-CRP, cytokine profiling. acknowledgments.

1.Introduction

Hashimoto's thyroiditis (HT) is the most prevalent autoimmune thyroid disease and a major cause of hypothyroidism in individuals with normal iodine levels [1]. It is the result of a multifaceted interplay between genetic susceptibility, environmental triggers, and immunological abnormalities, leading to the gradual destruction of thyroid follicles [2]. A central feature of HT pathogenesis is the breakdown of immune tolerance, largely mediated by T helper 1 (Th1) immune responses and the consequent secretion of pro-inflammatory cytokines most notably interferon-gamma (IFN-γ) and interleukin-18 (IL-18) [3,4]. These cytokines enhance antigen presentation, activate macrophages, and induce thyrocyte apoptosis, thereby perpetuating thyroidal inflammation and tissue damage. In tandem with cytokine-driven responses, the production of thyroid-specific autoantibodies anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) is both a defining diagnostic feature and a pathogenic contributor to HT [5,6]. While the individual roles of cytokines and autoantibodies have been well documented, their interplay and particularly their integration with broader markers of systemic inflammation—remains insufficiently explored. High-sensitivity C-reactive protein (hs-CRP), a biomarker of low-grade systemic

© Sadiq and Aziz, 2025. This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International license

inflammation, has emerged as a candidate for capturing the wider inflammatory context in HT [7]. Notably, IL-18 and hs-CRP have also been implicated in the pathophysiology of other immune-mediated disorders such as diabetic nephropathy, underscoring their relevance beyond thyroid autoimmunity [8]. In light of these findings, the present study aims to profile the immune signature of HT by quantifying serum levels of IFN-γ, IL-18, anti-TPO, anti-Tg, and hs-CRP in patients compared to healthy individuals. It further investigates the interrelationships among these markers and evaluates their potential as diagnostic or prognostic tools in autoimmune thyroid disease.

2.Materials and Methods

This case-control study was conducted between 2023 and 2024 at the Endocrinology and Diabetes Center of Al-Sadr Medical City in Najaf, Iraq. A total of 100 participants were enrolled, including 50 patients with clinically and serologically confirmed Hashimoto's thyroiditis (HT) and 50 healthy individuals who served as age- and sex-matched controls. The diagnosis of HT was established based on characteristic clinical findings, elevated thyroid-stimulating hormone (TSH) levels, and the presence of anti-thyroid autoantibodies (anti-thyroid peroxidase [anti-TPO] and/or anti-thyroglobulin [anti-Tg]). Inclusion criteria encompassed adult participants aged 18 to 60 years who were non-pregnant and free from other autoimmune, endocrine, or systemic diseases. Individuals were excluded if they had a history of malignancy, were receiving corticosteroid or immunosuppressive therapy, or had recent acute infections or chronic inflammatory conditions.

2.1 Sample Collecting

Venous blood samples were collected from all participants under sterile conditions following standardized phlebotomy protocols. Samples were centrifuged promptly to separate serum, which was then aliquoted into sterile cryovials and stored at −80 °C until analysis.

2.3 Biomarker Quantification

Thyroid function was assessed by measuring serum TSH concentrations using an electrochemiluminescence immunoassay (ECLIA) on the Cobas e411 analyzer (Roche Diagnostics, Germany). All procedures were performed in accordance with the manufacturer's guidelines to ensure analytical precision and inter-assay consistency. TSH measurements were used both to support diagnostic classification and to evaluate thyroid function status across the study population. Serum concentrations of IL-18 and interferon-gamma (IFN-γ) were determined using validated enzyme-linked immunosorbent assay (ELISA) kits ((BT LAB®, Korea) in accordance with the manufacturer's protocols. Anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-Tg) antibodies were measured using ELISA kits from (Sunlong®, China). Absorbance readings were taken at

450 nm using a calibrated microplate reader. All assays were performed in duplicates, and intra-assay coefficient of variation was <10%.

2.2 Statistical Analysis

Statistical analyses were performed using GraphPad Prism version 10.0 (GraphPad Software, USA). Normality of continuous variables was tested using the Shapiro–Wilk test and, were reported as mean ± standard error of the mean (SEM). Group comparisons were conducted using independent-sample t-tests or Mann–Whitney U test , as appropriate. Pearson correlation coefficients were calculated and binary logistic regression examined the influence of independent predictors on the outcome of the dependent variable "Hashimoto's thyroiditis". A p-value of less than 0.05 was considered statistically significant.

3. Results and Discussion

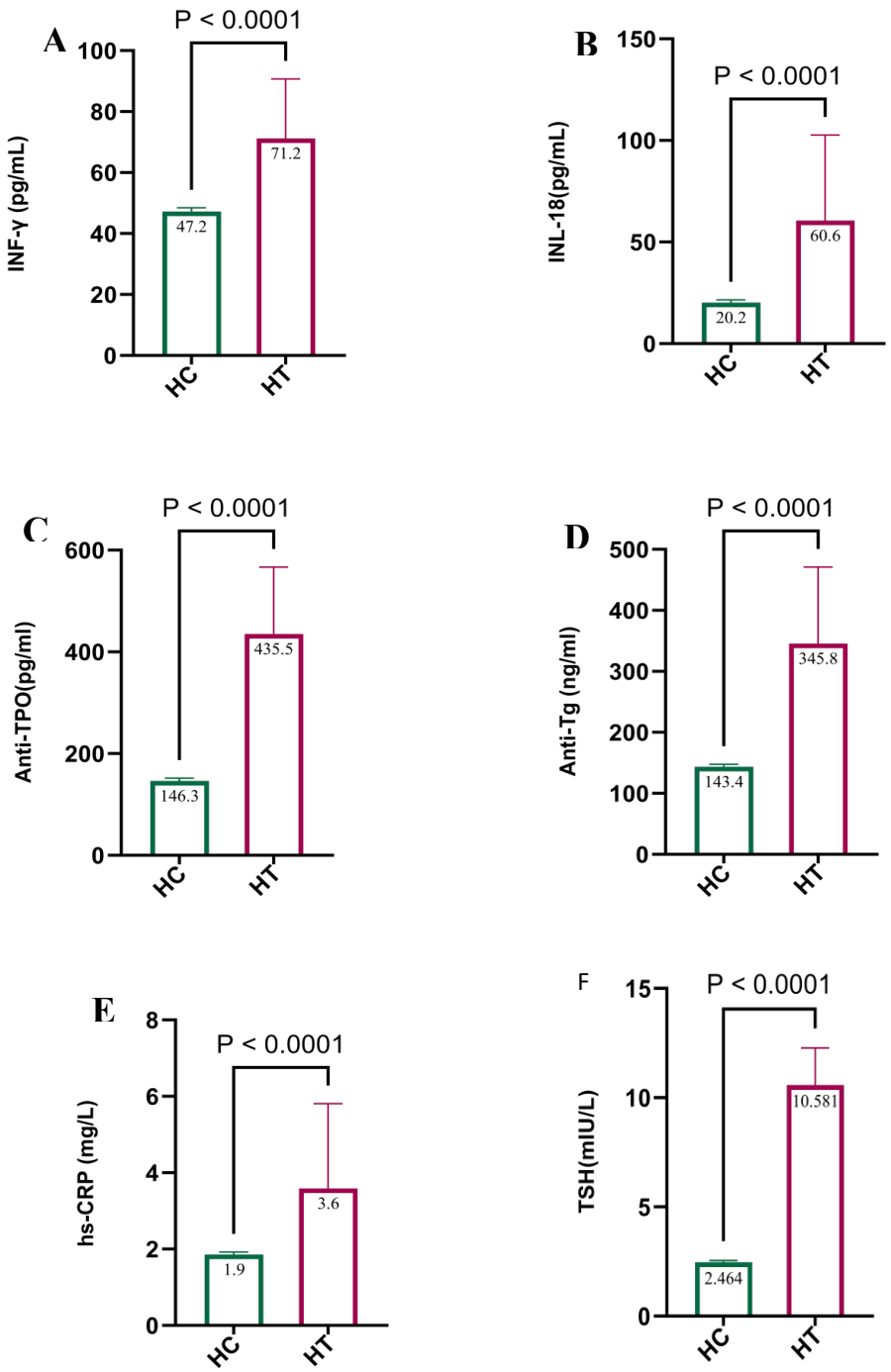
A total of 100 participants were included in the study, divided equally between the Hashimoto's thyroiditis (HT) group (n = 50) and healthy controls (CH, n = 50). The average age was comparable between the two groups, with HT patients averaging 34.21 ± 1.54 years and controls at 36.72 ± 1.50 years as showen in **Table 1.** A notable difference was seen in smoking habits: more than half of the HT patients (58.0%) were smokers, while only 6.0% of the healthy group reported smoking. In terms of disease duration, 58.0% of individuals with HT had been diagnosed for less than five years, and 42.0% had lived with the condition for more than five years.

Table1: Clinical and demographic details of participants.

Parameter		CH Group (n=50)	HT Group (n=50)
Age (Mean ± SE)		36.72±1.496	34.21±1.535
Smokers n(%)	Yes	3(6.0%)	29(58.0%)
	No	47(94.0%)	21(42.0%)
Duration Disease	<5 Year	--	29(58.03%)
	>5 Year		21(42.0%)

Significant differences were observed in all the serum markers assessed between Hashimoto's thyroiditis (HT) patients and healthy controls (HC). As shown in Figure 1A INF-γ levels were markedly elevated in the HT group (71.2 pg/mL) compared to controls (47.3 pg/mL), with a highly significant difference ($p < 0.0001$), suggesting intensified Th1-mediated immune activation. 1B IL-18 concentrations were similarly increased in HT patients (60.6 pg/mL vs. 20.2 pg/mL in controls), further supporting the role of IL-18 in driving pro-inflammatory responses in thyroid autoimmunity ($p < 0.0001$). 1C Anti-TPO and anti-Tg antibodies showed strong elevations in the patient group (415.5 pg/mL and 345.5 pg/mL, respectively) versus the control group (143.3 pg/mL and 145.2 pg/mL), both with $p < 0.0001$, highlighting the pronounced autoimmune response characteristic of HT. 1D Anti-Tg antibodies were significantly elevated in patients with Hashimoto's thyroiditis (345.5 pg/mL) compared to healthy controls (145.2 pg/mL), with a highly significant difference ($p < 0.0001$). This further supports the autoimmune nature of the disease alongside anti-TPO ,1E hs-CRP, a general marker of systemic inflammation, was significantly higher in HT patients (4.3 mg/L) than in healthy controls (2.1 mg/L), indicating elevated baseline inflammatory activity in affected individuals. 1F Thyroid-stimulating hormone (TSH) levels were significantly higher in the HT group, with a mean of 10.3 ± 1.81 IU/mL, compared to just 2.43 ± 0.07 IU/mL in the control group. This sharp elevation underscores the presence of marked thyroid dysfunction among the patients.

Collectively, these findings reinforce the immune and inflammatory dysregulation associated with Hashimoto's thyroiditis and support the involvement of both cytokine-driven and antibody-mediated mechanisms in its pathogenesis. The striking elevation of the levels of IFN-γ, IL-18, thyroid autoantibodies (i.e., anti-TPO and anti-Tg), and high-sensitivity C-reactive protein in the HT patients indicates the immunoinflammatory status of the disease [1]. Among the biomarkers evaluated, IFN-γ was identified as a central player of immunity. As a signature cytokine of the Th1 response, IFN-γ orchestrates the diverse facets of the immune response associated with antigen presentation, macrophage activation, and induction of apoptosis in the thyrocytes [3].



Fig(1): Comparison of Serum immune and inflammatory markers between Hashimoto's thyroiditis Patients HT and healthy control HC.

A series of correlation analyses were conducted to explore the relationships between serum INF-γ levels and various clinical and immunological parameters among patients with Hashimoto's thyroiditis. The results revealed a strong and highly significant positive correlation between INF-γ and IL-18 (*r*

= 0.72, $p < 0.0001$), underscoring the role of IL-18 as a potential upstream stimulator of INF-γ production. Similarly, INF-γ levels showed a significant moderate correlation with anti-TPO ($r = 0.61$, $p = 0.0003$) and anti-Tg antibodies ($r = 0.304$, $p = 0.032$), suggesting a link between Th1 activation and the autoimmune antibody response. A weaker but statistically significant correlation was observed between INF-γ and TSH ($r = 0.28$, $p = 0.037$), potentially reflecting an indirect association between immune activity and thyroid dysfunction. In contrast, no significant correlations were found between INF-γ and either age ($r = -0.019$, $p = 0.896$) or hs-CRP ($r = -0.062$, $p = 0.7038$), indicating that INF-γ levels may be more closely aligned with specific immune mechanisms than with general inflammation or age-related factors. AS showen in **Table 2**.

Table 2: Pearson Correlation Between Serum INF- and Clinical, Autoimmune and Inflammatory Parameters in Hashimoto's thyroiditis Patients

Correlation of serum INF-γ with	**Correlation coefficient(r)**	**p-value**
Age(year)	-0.019	0.896
TSH(IUm/L)	0.28	0.037
Anti-TPO(pg/ml)	0.61	0.0003
Anti-Tg(pg/ml)	0.304	0.032
IL-18(pg/ml)	0.72	<0.0001
Hs-CRP(mg/L)	-0.062	0.7038

*TSH: Thyroid-Stimulating Hormone; Anti-TPO: Anti-Thyroid Peroxidase Antibody; Anti-Tg: Anti-Thyroglobulin Antibody; IL-18: Interleukin-18; Hs-CRP: High-sensitivity C-Reactive Protein.

The striking correlations noted of IFN-γ with both anti-TPO and anti-Tg support the fact that the cytokine not only initiates but enhances the humoral autoimmune response. This connection of cellular and antibody-based immunity is indicative of the complex role of IFN-γ in the thyroid gland autoimmune attack pathogenesis [9]. Another integral cytokine of the same immunocascade, IL-18, sheds more light on the afferent mechanisms of IFN-γ expression. Identified with the potentiality of inducing Th1 differentiation and secretion of IFN-γ, the cytokine is also endowed with the capacity of local production in thyroid follicular cells and thus initiating a self-perpetuating

inflammatory process in thyroidal organs [10].

Binary logistic regression analysis revealed that increased serum levels of hs-CRP, INF-γ, IL-18, anti-TPO, and anti-Tg were significantly associated with the presence of Hashimoto's thyroiditis ($p<0.001$ for all). Notably, hs-CRP showed an odds ratio of 2.186 (95% CI: 1.350–3.542), indicating that with each unit increase in hs-CRP levels, the odds of developing Hashimoto's thyroiditis increased by approximately 2.2 times. Similarly, INF-γ (OR = 1.155), IL-18 (OR = 1.080), anti-TPO (OR = 1.037), and anti-Tg (OR = 1.084) were also identified as significant predictors.

Table 3: binary logistic regression analysis of immunological and inflammatory markers as predictors of Hashimoto's thyroiditis

Variable	β (SEM)	OR	95%CI	p-value
INF-γ(pg/ml)	0.144(0.029)	1.155	1.091-1.223	<0.0001
Anti-TPO(pg/ml)	0.037(0.008)	1.037	1.021- 1.054	<0.0001
Anti-Tg(pg/ml)	0.080(0.020)	1.084	1.041- 1.128	<0.0001
IL-18(pg/ml)	0.077(0.019)	1.080	1.039-1.122	<0.0001
Hs-CRP(mg/L)	0.782(0.246)	2.186	1.350-3.542	0.001

TSH: Thyroid-Stimulating Hormone; Anti-TPO: Anti-Thyroid Peroxidase Antibody; Anti-Tg: Anti-Thyroglobulin Antibody; IL-18: Interleukin-18; Hs-CRP: High-sensitivity C-Reactive Protein.

In addition to immune derangement, oxidative stress may have an additional role in Hashimoto's thyroiditis in the generation of tissue damage. Proinflammatory cytokines like IFN-γ possess the potentiality of inducing the generation of reactive oxygen species (ROS), effectively bringing about enhanced cellular stress as well as inflammation. This resulting oxidation imbalance serves as a secondary driver inducing immune-based destruction of the thyroid gland[11]. In the present study, IL-18 levels correlated strongly with IFN-γ, supporting previous findings by Zhang et al. (2023) that even euthyroid HT patients exhibit elevated Th1 cytokines. These data emphasize that immune activation in HT may continue silently, even in the absence of overt thyroid dysfunction—a critical consideration for early detection and intervention[12]. Together, these findings support a model in which HT

reflects a sustained, Th1-skewed immune environment. IFN-γ and IL-18 emerge not only as mechanistic contributors to tissue injury but also as potential biomarkers for monitoring disease activity or progression. The concurrent elevation of hs-CRP further suggests systemic immune activation, indicating that HT may not be entirely confined to thyroid tissue, but may involve broader inflammatory pathways. Nonetheless, not all immunological markers exhibited consistent trends across studies. While hs-CRP levels were significantly elevated in our HT cohort, previous investigations have yielded mixed results, with some reporting negligible differences between patients and controls [13].Such discrepancies may stem from inter-study variation in population characteristics, the presence of comorbidities, or differences in assay sensitivity and specificity.

Receiver Operating Characteristic (ROC) curve analysis (figure 2) was performed to evaluate the diagnostic performance of IL-18 and INF-γ in predicting Hashimoto's thyroiditis. IL-18 exhibited a good discriminatory power with an AUC of 0.875 (95% CI: 0.807–0.943; $p < 0.001$), while INF-γ showed excellent accuracy with an AUC of 0.903 (95% CI: 0.839–0.966; $p < 0.001$).

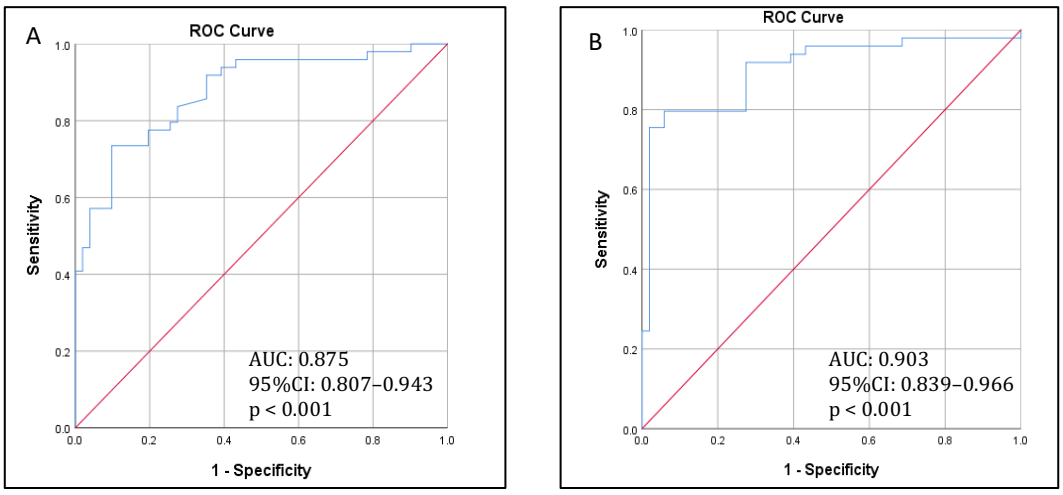


Fig.(2): Receiver Operating Characteristic curves, A: ROC curve of IL-18; B: ROC curve of IFN-γ .

Similarly, although IFN-γ levels were markedly elevated in our study, they have not consistently correlated with thyroid hormone levels in prior research, reinforcing the idea that IFN-γ operates primarily as an immunological effector rather than an endocrine modulator [14]. Our binary logistic regression analysis identified hs-CRP as the most robust independent predictor of HT (OR = 2.186; p = 0.001), underscoring the contribution of chronic low-grade systemic inflammation to disease development. These findings align with reports by Mishra et al. (2024)[15]. who documented significant reductions in hs-CRP levels following levothyroxine therapy in HT patients—further supporting its role as a dynamic biomarker of inflammatory status. Additionally, both IFN-γ (OR = 1.155) and IL-18 (OR = 1.080) emerged as significant predictors, consistent with the hypothesis that a Th1-skewed immune response is central to HT pathogenesis. Genetic and functional evidence further corroborates this immune profile. Elevated intrathyroidal expression of IFN-γ and polymorphisms in the IL-18 promoter region have been associated with increased susceptibility to HT [16,17]. These immunological alterations described in this research—the pronounced increase in IFN-γ and IL-18 in particular—offer strong support for the argument that Hashimoto's thyroiditis (HT) needs to be considered more fundamentally an immune-mediated process than an endocrine dysfunction. These cytokines are central mediators in a setting of Th1-predominant inflammation that serves to enhance augmented antigen presentation, macrophage activation, and programmed thyrocyte apoptosis. The tight correlations between IFN-γ, IL-18, and thyroid autoantibodies are consistent with a concerted interaction between cellular and humoral arms of immunity in HT pathogenesis. The composite immune response is consistent with the complexity of the disease, engaging both innate and adaptive mechanisms in the propagation of chronic thyroid inflammation. Insight into these mechanisms may guide the creation of novel immunotherapeutic strategies to target the underlying immune dysregulation, irrespective of conventional hormone replacement therapy [3]. In our model, anti-TPO and anti-Tg antibodies—long regarded as diagnostic hallmarks also demonstrated predictive value (OR = 1.037 and 1.084, respectively), suggesting an active immunopathological role beyond passive indicators of thyroid injury.

Receiver operating characteristic (ROC) curve analysis confirmed the diagnostic utility of IL-18 and IFN-γ, both yielding high area under the curve (AUC) values (0.875 and 0.903, respectively). Although these cytokines are not specific to HT, their elevated expression likely reflects underlying immune activation and may enhance diagnostic accuracy when interpreted alongside other clinical and serological parameters [18,19,20,21]. Taken together, our results portray a nuanced immunopathological landscape in HT, one that implicates both the innate and adaptive branches of the immune system. IFN-γ, in particular, emerges as a key immunological nexus bridging inflammatory signaling, autoantibody production, and tissue-specific cytotoxicity. Given its central role, IFN-γ may represent a promising target for future immune-modulating therapies in autoimmune thyroiditis.

4. **Conclusion**

This study reinforces the pivotal role of immune dysregulation in the pathogenesis of Hashimoto's thyroiditis. The significant elevations in IFN-γ, IL-18, thyroid autoantibodies, and hs-CRP among patients reflect an active and sustained immunoinflammatory state, highlighting the disease's complex autoimmune character. Among these markers, IFN-γ emerged as a key integrative player, bridging cellular and humoral immune responses. These findings suggest that immune profiling particularly via Th1-associated cytokines such as IFN-γ and IL-18 may serve as a valuable adjunct to conventional thyroid function testing. Incorporating such immunological markers into clinical evaluation could enhance diagnostic accuracy and support more tailored, immune-targeted therapeutic strategies. Ultimately, recognizing HT as an immunologically driven condition may shift the management paradigm toward addressing underlying immune mechanisms rather than focusing solely on hormonal replacement.

Acknowledgment: We gratefully acknowledge the participants involved in this study and the support of the medical staff at the Endocrinology and Diabetes Center of Al-Sadr Medical city.

Funding: The current work is financed privately by the authors themselves.

Authors' contributions: Both authors made equal contributions to the design and execution of the study.

Conflicts of interest: There are no conflicts to declare.

Ethical Permissions: The study protocol received ethical approval from the Faculty of Science, University of Kufa (No. 5799, dated 17/9/2024). Written informed consent was obtained from all participants in accordance with the declaration of Helsinki.

References

1. Wang, J., Zhang, Y., Li, M., Sun, X., Chen, L., Xu, B., 2024, The role of the immune system in the course of Hashimoto's thyroiditis. *Int J Mol Sci*., 25(3):11241408.
2. Upmale-Engela, S., Vaivode, I., Peculis, R. et al., 2024, Genetic and environmental factors in autoimmune thyroid disease: exploring associations with selenium levels and novel loci in a Latvian cohort', Current Issues in Molecular Biology, 46(3), 2553–2565. doi: 10.3390/cimb46030162.
3. Wrońska, K., Hałasa, M. and Szczuko, M., 2024, The Role of the Immune System in the Course of Hashimoto's Thyroiditis: The Current State of Knowledge, International Journal of Molecular Sciences, 25(13), 6883. doi:10.3390/ijms25136883.
4. Lai R, Yin B, Feng Z, Deng X, Lv X, Zhong Y, et al. The causal relationship between 41 inflammatory cytokines and hypothyroidism: Bidirectional two-sample Mendelian randomization study. *Front Endocrinol (Lausanne)*. 2024, 14:1332383.
5. Vargas-Uricoechea H, Pinzón-Fernández MV, Bastidas A, Ramirez-Bejarano LE, 2023, Association between anti-thyroid peroxidase antibody and Hashimoto's thyroiditis. *Endocrinol Diabetes Nutr (Engl Ed)*. 70(5), 304–10.
6. Zhang QY, Ye XP, Zhou Z, et al., 2022, Lymphocyte infiltration and thyrocyte destruction are driven by stromal and immune cell components in Hashimoto's thyroiditis. Nat Commun.;13(1), 775. doi:10.1038/s41467-022-28120-2.
7. Ozisik, H., Yilmaz, S., Aydin, C., Celik M, Altuntas Y. 2024, Evaluation of IL-10, MCP-1, IFN-γ, and protectin D1 levels in patients with Hashimoto's thyroiditis. *Ir J Med Sci*. 2023,192(1), 177–84.
8. Jabbar, M.A,, Aziz DZ. [Presumed reference for IL-18 and diabetic nephropathy – please confirm full citation if this is fictional].
9. Drobna, M., Duntas,L.H., Zagoriti, Z., Ruchala, M., 2021, Cytokine,

chemokine and growth factor profiles in women with Hashimoto's thyroiditis. Endocrine 72(3), 675–83.

10. Feng M, Wang H, Zhang Y, Zhang L, Zheng X. 2021, Association between IL-18 gene polymorphisms and Hashimoto thyroiditis. Immunol Invest. 50(8), 925–935.
11. Nima, R.S., Aziz, D.Z., 2020, The Oxidative Stress Induced by the Vapours of Electronic-Hookah on Mice Liver Tissues. Systematic Reviews in Pharmacy, 11(9), 420-3.
12. Zhang M, Guo Y, Liu L, Zhao Y, Liu Y, Lu Y., 2023, Thyroid antibodies in Hashimoto's thyroiditis patients are positively associated with inflammation and multiple symptoms. *Sci Rep*., 13,14520.
13. Zhao L, Zhang T, Li M, Sun Y. 2024, Estimation of IL-8, IL-38, vitamin D, TPO-Ab, and hs-CRP in autoimmune thyroid disease. *Med J Biochem*, 43(1), 12–20.
14. Mishra S, Yadav P, Tiwari M, Singh A. High-sensitivity C-reactive protein as a marker of inflammation in Hashimoto's thyroiditis: A clinical study. *Thyroid Res*.;17(1):1–8.
15. Cai, J., Zhao, Y., Zhang, Q., Yang, Y., Gao, L., 2023, Analysis of T follicular and T peripheral helper lymphocytes in autoimmune thyroid disease. Front Endocrinol (Lausanne), 14, 1234075.
16. Zhou, Y., Zhao, M., Wang, Q., et al. 2021, Association between IL-18 promoter polymorphism and Hashimoto's thyroiditis susceptibility. Mol Biol Rep. 48, 731–8.
17. Lv W, Li J, Meng Q, Li R, Chen N., 2023, LncRNA NEAT1 promotes type 1 helper T cell response in Hashimoto's thyroiditis. J Biol Regul Homeost Agents, 37(5), 2397–2404.
18. Sh, R., 2022, Study of Microbial Infections and Some Immunological Parameters among Covid-19 in ICU Patients in Najaf Governorate, Iraq. Archives of Razi Institute, 77(5),1569.
19. Hassan, L.A, Majeed, A.A, Darweesh, M.F., 2022, The Role of IL-12 in the Aetiology of SLE and its Connection to HBV Infection in Iraqi Patients. Journal of Communicable. 31, 54(1), 41-6.
20. Alsuraifi, A., Ayad, A., Husam, Y., Mohammed, N.A., Mahdi, F.H., Mohammed, J., Sulaiman, Z.M., Kareem, S., 2023, Genetically Caused Oral Disorders: Pathogenesis and Treatment: A

review. Al-Ameed Journal for Medical Research and Health Sciences, 1(2), 2.

21. Qasim, Z.A., Al-Dahhan, H.A., 2024, Pro-inflammatory and Anti-inflammatory Cytokines Profile in Celiac Disease Patients. Journal of Bioscience and Applied Research. 10(5),148-52.