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Inflammatory biomarkers in Hashimoto's thyroiditis: a comparative cross-sectional study

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ABSTRACT

Aims: Hashimoto's thyroiditis (HT) is an autoimmune disorder impacting majorly females. HT is asymptomatic long-term and known to be affected by metabolic factors, genetics, and inflammation. The study aimed to compare inflammatory and metabolic markers in HT and assess their value in the diagnosis.

Methods: An HT group (n=103) and a control group with euthyroidism (n=103) were included in the study. The demographics, anthropometric measurements, and circulation-based inflammatory and metabolic markers, triglyceride-glucose index, FIB-4 score, non-alcoholic fatty liver disease score, APRI, ATH score, systemic immune-inflammation index, systemic inflammation response index, prognostic nutritional index, monocyte-to-HDL cholesterol ratio, atherogenic index of plasma, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, PV, PDW, and RDW were recorded.

Results: Consistent with the literature, we found a 10-fold difference in the HT prevalence in females (90.3%) compared with males (9.7%). Anthropometric measurements revealed that increased hip circumference increases the risk of HT (118.0 cm [107.0–132.0]) compared to control (110.0 cm [103.0–119.0]). We failed to find any difference in metabolic or inflammatory indices in the HT vs. control group.

Conclusion: This study reinforces the well-documented association between HT and female predominance and metabolic factors such as obesity and diabetes. However, inflammatory markers did not show a significant association with HT, urging the need for larger cohorts.

Keywords: Hashimoto thyroiditis, inflammatory markers, hip circumference, metabolic markers

INTRODUCTION

Hashimoto's thyroiditis (HT) is an autoimmune condition that arises when the body's immune system fails to tolerate key thyroid-specific proteins—thyroglobulin (TG) and thyroid peroxidase (TPO). This loss of tolerance leads to chronic inflammation and gradual destruction of the thyroid gland.[1-3] HT is one of the most common endocrine disorders, affecting about 7.5% of the global population, with a notably higher prevalence in women.[4] It also increases the risk of certain thyroid cancers, particularly papillary thyroid carcinoma.[1,5]

Both genetic predisposition and environmental exposures—along with metabolic comorbidities such as obesity and diabetes—are believed to contribute to HT development.[2,3,6] The underlying autoimmune activity is often driven by immune cell infiltration and the formation of structures known as tertiary lymphoid organs within the thyroid tissue.

Recently, there has been growing interest in whether certain blood-based inflammatory markers—including the neutrophil-to-lymphocyte ratio (NLR),[7-9] platelet-to-lymphocyte ratio (PLR),[8,10,11] and systemic immune-inflammation index (SII)[9,11]-might help identify disease severity or progression. However, findings remain inconsistent. This study aimed to explore these markers in patients with HT and compare them with healthy, euthyroid individuals to better understand their potential diagnostic value.

METHODS

Ethics

This cross-sectional comparative study was conducted with patients who were diagnosed with HT previously, and a control group consisted of patients referred to the endocrinology clinic with symptoms related to a possible thyroid pathology between October 2021 and August 2023. Necessary ethical compliance and approvals were granted by the Karabük University Non-interventional Clinical Researches Ethics Committee (Date: 06.01.2025, Decision No: 2025/2055). The study was carried out according to the Declaration of Helsinki, and the strengthening the reporting of observational studies in epidemiology (STROBE) guidelines[12] were implemented.

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Written consent was obtained from the patient participating in this study.

Study Population

The inclusion criteria of the HT group were: (a) being 18 years and older, (b) holding a prior HT diagnosis, (c) having a heterogeneous thyroid pattern in USG, and (d) anti-TG and anti-TPO larger than 4.5 U/ml and 60 U/ml. For the control group, adults who were referred to the department with a possible thyroid disease with a biochemical euthyroid, homogenous thyroid in USG, and have anti-TG lower than 4.5 U/ml and anti-TPO lower than 60 U/ml were included. Within the HT study group patients who (a) do not have heterogenous thyroid in USG, who have (b) anti-TG lower than 4.5 U/ml or anti-TPO U/ml lower than 60 U/ml, (c) hepatic, renal and/or heart failure, (d) diabetes mellitus, (e) any active malignancy, (f) history of organ transplantation, (g) pregnancy, (h) chronic inflammatory disease, (i) anemia or polycythemia, (j) acute or chronic infection, (k) lipid-lowering or hepatotoxic medication use, (l) received medications affecting CBC, (m) alcohol and tobacco use, (n) major surgery within the last six months were excluded. For the control group with a possible thyroid disease the patients who (a) are not biochemically euthyroid, (b) do not have homogenous thyroid in USG, who have (c) anti-TG larger than 4.5 U/ml or anti-TPO larger than 60 U/ml, (d) hepatic, renal and/or heart failure, (e) diabetes mellitus, (f) any active malignancy, (g) history of organ transplantation, (h) pregnancy, (i) chronic inflammatory disease, (j) anemia or polycythemia, (k) acute or chronic infection, (l) lipid-lowering or hepatotoxic medication use, (m) received medications affecting CBC, (n) alcohol and tobacco use, (o) major surgery within the last six months were excluded.

Data and Variables

The HT diagnosis of the patients was confirmed according to previous guidelines (Caturegli 2014). In this study, we examined a range of metabolic and inflammatory markers. These included the triglyceride-glucose (TyG) index, non-alcoholic fatty liver disease (NAFLD) score, and prognostic nutritional index (PNI), along with SII, systemic inflammation response index (SIRI), monocyte-to-HDL cholesterol ratio (MHR), atherogenic index of plasma (AIP), and the NLR and PLR ratios. Each of these markers has been proposed in earlier research as a potential indicator of metabolic or immune activity. The age, gender, body-mass index (BMI), anthropometric, and laboratory measurements were recorded.

Weight was measured using a digital scale (Omron, Japan), and height was recorded with a stadiometer. BMI was then calculated.

$$BMI=\text{weight [kg]}/\text{height [m]}^2$$

Waist circumference (WC) was assessed using a measuring tape while subjects stood in a standard position—with feet together, arms at their sides, and an unflexed abdomen—with the measurement taken between the subcostal plane and the iliac crest. Hip circumference (HC) was measured at the point of maximum hip girth, and the waist-to-hip ratio (WHR) was subsequently calculated by dividing WC (cm) by HC (cm).

Laboratory evaluations including triglycerides, free T3 and T4, TSH, TG, anti-TG, anti-TPO, calcitonin, liver enzymes, full hemogram panel, and serum albumin were measured from blood samples. Blood draws were generally performed in the morning, ensuring an 8-hour fasting period. The TyG was calculated using the formula:

$$TyG=Ln(\text{(fasting triglycerides [mg/dl]}\times\text{fasting blood glucose [mg/dl])}/2)$$

The FIB-4 score is calculated to assess liver fibrosis according to

$$\text{FIB-4 score}=(\text{age (years)}\times\text{AST (U/L)})/(\text{Platelet }(10^9/L)\times\sqrt{}\text{ALT (U/L)})$$

NAFLD score assists the severity of liver fibrosis; mild (F1-F2) vs. advanced (F3-F4) and calculated as follows:

$$\text{NAFLD score}=-1.675+0.037\times\text{age (years)}+0.094\times\text{BMI (kg/m}^2)+1.13\times(\text{IFG/DM [yes=1, no=0]})+0.99\times(\text{AST/ALT})-0.013\times\text{platelets }(10^9/L)-0.66\times\text{albumin (g/dl)}$$

The aspartate-to-platelet ratio (ASPI) score measures the likelihood of hepatic fibrosis and cirrhosis according to the formulae below:

$$\text{APRI score}=[(\text{AST/ULN})\times100]/\text{platelet }(10^9/L)$$

The PNI determines the balance between albumin and immunological cell accumulation,

$$PNI=10\times\text{serum albumin (g/dl)}+0.005\times\text{total lymphocyte count (/mm}^3)$$

The inflammatory markers, SII, SIRI, MHR, AIP, PLR and NLR were calculated accordingly:

$$SIRI=(\text{neutrophil}\times\text{monocyte})/\text{lymphocyte}$$

$$MHR=\text{monocyte/HDL}$$

$$AIP=Ln\ [TG/HDL]$$

$$NLR=\text{neutrophil/lymphocyte}$$

$$PLR=\text{platelet/lymphocyte}$$

Statistical Analysis

No prior sample size was calculated because we intended to include all eligible patients in the study. Statistical analyses were performed using SPSS version 23 (IBM Corp. in Armonk, NY). The distributions of numerical variables were evaluated using the Shapiro-Wilk and Kolmogorov-Smirnov tests. Descriptive statistics were presented as frequency (n) and percentage (%) for categorical variables, and median with an interquartile range (IQR) for non-normally distributed numerical variables. Numerical data were analyzed using the Mann-Whitney U test to compare differences between the HT group and the control group. Categorical data were analyzed using the Pearson Chi-square test or Fisher's exact test. $p<0.05$ was accepted as the statistical significance level.

RESULTS

A total of 823 patients were assessed for eligibility, including 249 patients with a prior diagnosis of HT and 574 individuals presenting symptoms of a possible thyroid disease. HT patient; without a heterogenous thyroid in USG (n=8), have

an anti-TG or anti-TGO levels lower than 4.5 U/ml and 60 U/ml (n=14), and have hepatic (n=2), renal (n=5), heart (n=3) failure, diabetes (n=28), any other active malignancy (n=4), history of solid organ transplantation (n=3), are pregnant (n=2), have chronic inflammatory diseases (n=7), have any anemia or polycythemia (n=23), acute or chronic infection (n=6), who are taking lipid-lowering (n=14) or hepatotoxic (n=5) medication or taking any medication affecting their CBC (n=2), using alcohol and tobacco (n=19) and who had major surgery within the past six months (n=1) were excluded from the study (n=146). Within the control group, patients who are not biochemically euthyroid (n=43), do not have homogenous thyroid in USG (n=26), have an anti-TG level larger than 4.5 U/ml or anti-TGO levels larger than 60 U/ml (n=39), who have hepatic (n=3), renal (n=11), heart (n=4) failure, diabetes (n=77), any other active malignancy (n=6), history of solid organ transplantation (n=3), are pregnant (n=7), have chronic inflammatory diseases (n=12), have any anemia or polycythemia (n=32), acute or chronic infection (n=15), who are taking lipid-lowering (n=31) or hepatotoxic (n=11) medication or taking any medication affecting their CBC (n=9), using alcohol and tobacco (n=33) and who had major surgery within the past six months (n=7) were excluded from the study (n=369). Of the eligible patients for the control group (n=205), 103 were randomly selected and allocated to the control group. All eligible patients for the HT group (n=103) were included in the HT group. Totally, the data of 206 patients were analyzed (**Figure**).

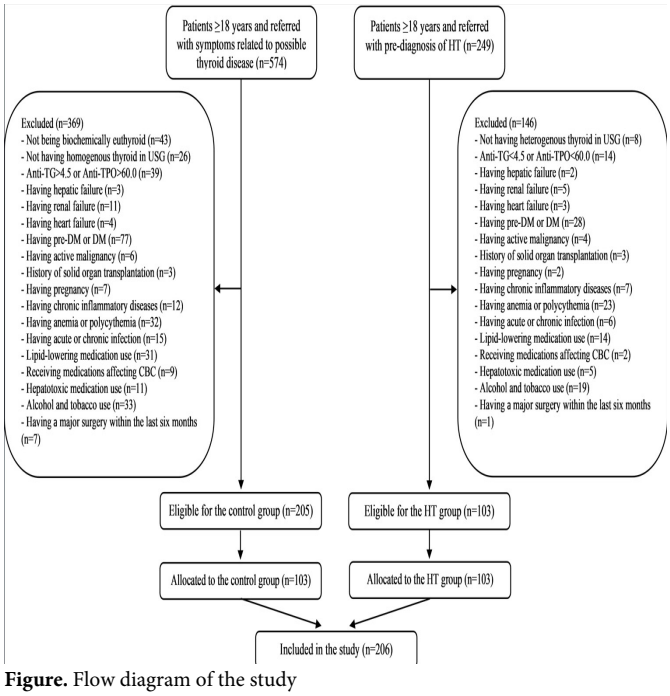

Figure. Flow diagram of the study

Demographic and clinical findings are presented in **Table 1**. Although the groups were comparable in terms of age, BMI, and WC, the HT group had a significantly increased proportion of females (90.3% vs. 76.7%, p=0.009) and a larger HC (median 118.0 cm vs. 110.0 cm, p<0.001). The thyroid function was uniformly distributed within the control group, all presented with euthyroid (n=103, 100%), whereas in the study group, only 57.3% of the HT group were euthyroid, 34% were hypothyroidism, and the rest 8.7% presented with subclinical hypothyroidism (p<0.001). Free T3 and T4 levels were comparable between groups; however, the HT group had significantly higher TSH levels (median 3.45 vs. 1.70 μIU/ml, p<0.001), elevated anti-TG (7.00 vs. 0.10 IU/ml, p<0.001) and anti-TPO levels (782.0 vs. 35.8 IU/ml, p<0.001), along with a significantly lower TG concentration (11.4 vs. 24.3 ng/ml, p<0.001). We also found a moderate positive correlation between anti-TG antibody levels and TG concentration (r=0.548, p<0.001), indicating that patients with measurable TG levels often had correspondingly elevated anti-TG antibodies. No significant differences were observed in terms of family history of thyroid cancer, history of neck radiotherapy, or calcitonin levels (**Table 1**).

Table 2 compares various inflammatory markers between the HT and control groups. No statistically significant differences were observed in any of the parameters, including the TyG index, FIB-4 score, NAFLD score, APRI, ATH score, SII, SIRI, PNI, MHR, AIP, NLR, PLR, MPV, PDW, and RDW (all p>0.05), indicating that the inflammatory profiles are similar between the two groups (**Table 2**).

DISCUSSION

This cross-sectional study compares the inflammatory biomarkers between patients with HT and a non-HT euthyroid control group. We found that despite the differences in thyroid function and autoantibody levels, the two groups did not significantly differ in various inflammatory indices, including hepatic and cardiometabolic markers. The gender difference in HT is well documented.[2,13] Studies have shown that females are more commonly impacted, and the female-to-male ratio is 7-10:1.[2,14] Similar to the literature, our findings show a 10-fold gender difference, with a pronounced female predominance. The disparity is mainly attributed to hormonal differences, specifically the imbalance between estradiol and progesterone in patients with polycystic ovary syndrome,[15] increased concentrations of estradiol, and reduced levels of testosterone in female HT patients.[16] However, studies also show that sex hormones are not the only culprit.[17] Increased BMI, signs of metabolic obesity, and WC correlated with the risk of HT.[18] Another study found that gender introduces differential fat distribution; within the same BMI range, females possess more adiposity, indirectly indicating a more insulin-sensitive environment, predisposing female diabetics to HT.[19] Our findings presented that increased HC (median in men 107.0 cm [IQR:101.2-110.0], and median HC in women 115.0 cm [IQR:106.0-125.0]) was associated with HT risk. Recent studies also have shown that changing diet to a gluten-free, Mediterranean diet[20,21] supplemented with vitamin D[22] improves anthropometric measures and regulates TSH levels. This link between TSH levels and anthropometric measurements is evident in our cohort, where the median HC was 110.5 cm (IQR:104.0-122.3) in euthyroid, 124.0 cm (IQR:116.5-140.0) in subclinical hypothyroid, and 120.0 cm (IQR:107.0-133.0) in hypothyroid HT patients (data not shown).

Our results emphasize the heterogeneous nature of thyroid function among patients with HT. While a substantial proportion of HT patients (57.3%) remained euthyroid,

Table 1. Demographics and laboratory findings of Hashimoto's thyroiditis and the control groups

Variables	Control group (n=103)	HT group (n=103)	p
Age (year), median (IQR)	50.0 (47.0-57.0)	51.0 (43.0-59.0)	0.810[a]
Gender, n (%)			0.009[b]
Male	24 (23.3)	10 (9.7)	
Female	79 (76.7)	93 (90.3)	
Family history of thyroid cancer, n (%)	5 (4.9)	3 (2.9)	0.721[c]
History of neck radiotherapy, n (%)	3 (2.9)	2 (1.9)	0.999[c]
BMI (kg/m²), median (IQR)	29.5 (26.7-33.0)	28.5 (24.5-33.5)	0.304[a]
WC (cm), median IQR)	100.0 (94.0-109.0)	102.0 (90.0-115.0)	0.729[a]
HC (cm), median (IQR)	110.0 (103.0-119.0)	118.0 (107.0-132.0)	<0.001[a]
Thyroid function status, n (%)			<0.001[c]
Hypothyroidism	0 (0.0)	35 (34.0)	
Subclinical hypothyroidism	0 (0.0)	9 (8.7)	
Euthyroidism	103 (100.0)	59 (57.3)	
Free T_3 (pg/ml), median (IQR)	3.33 (3.09-3.58)	3.29 (2.97-3.47)	0.139[a]
Free T_4 (pg/ml), median (IQR)	1.24 (1.10-1.38)	1.21 (1.06-1.34)	0.116[a]
TSH (µIU/ml), median (IQR)	1.70 (0.96-2.65)	3.45 (1.50-4.62)	<0.001[a]
Thyroglobulin (ng/ml), median (IQR)	24.3 (11.6-62.8)	11.4 (1.47-40.2)	<0.001[a]
Anti-TG (IU/ml), median (IQR)	0.10 (0.00-0.50)	7.00 (1.10-73.00)	<0.001[a]
Anti-TPO (IU/ml), median (IQR)	35.8 (28.0-46.2)	782.0 (131.0-1300.0)	<0.001[a]
Calcitonin, n (%)			0.999[c]
Normal	101 (98.1)	102 (99.0)	
High	2 (1.9)	1 (1.0)	

[a]Mann-Whitney U Test was used. [b]Pearson Chi-square Test was used. [c]Fisher's Exact Test was used. HT: Hashimoto's thyroiditis, IQR: Inter-quartile range, BMI: Body-mass index, WC: Waist circumference, HC: Hip circumference, T_3: Triiodothyronine, T_4: Thyroxine, TSH: Thyroid-stimulating hormone, Anti-TG: Anti-thyroglobulin antibody, Anti-TPO: Anti-thyroid peroxidase antibody

Table 2. Comparison of inflammation markers between Hashimoto's thyroiditis and control groups

Parameters	Control group (n=103)	HT group (n=103)	p
TyG index, median (IQR)	4.66 (4.44-4.85)	4.70 (4.47-4.86)	0.526[a]
FIB-4 score, median (IQR)	0.86 (0.65-1.12)	0.85 (0.55-1.13)	0.682[a]
NAFLD score, median (IQR)	-2.27 (-2.91--1.57)	-2.41 (-3.27--1.62)	0.395[a]
APRI, median (IQR)	0.20 (0.15-0.27)	0.21 (0.15-0.26)	0.918[a]
ATH score, median (IQR)	0.39 (0.15-0.57)	0.37 (0.21-0.56)	0.916[a]
SII, median (IQR)	488.7 (358.3-717.5)	581.0 (396.0-822.9)	0.121[a]
SIRI, median (IQR)	0.72 (0.53-1.01)	0.82 (0.57-1.25)	0.116[a]
PNI, median (IQR)	54.5 (52.0-57.0)	55.0 (52.5-58.0)	0.492[a]
MHR, median (IQR)	7.35 (5.75-10.25)	8.37 (5.85-10.73)	0.411[a]
AIP, median (IQR)	0.39 (0.15-0.57)	0.37 (0.21-0.56)	0.916[a]
NLR, median (IQR)	1.89 (1.42-2.47)	1.94 (1.56-2.65)	0.280[a]
PLR, median (IQR)	130.8 (103.5-170.0)	136.0 (108.7-169.3)	0.462[a]
MPV, median (IQR)	10.2 (9.5-10.9)	10.2 (9.7-11.0)	0.350[a]
PDW, median (IQR)	16.1 (15.9-16.3)	16.1 (15.9-16.3)	0.770[a]
RDW, median (IQR)	42.7 (41.0-44.8)	42.9 (40.9-45.2)	0.872[a]

[a]Mann-Whitney U Test was used. TyG: Triglyceride-glucose index, FIB-4 score: Fibrosis-4 score, SIRI: Systemic inflammation response index, PNI: Prognostic nutritional index, MHR: Monocyte-to-high-density lipoprotein cholesterol ratio, AIP: Atherogenic index of plasma, PLR: Platelet-to-lymphocyte ratio, PDW: Platelet distribution width, SII: Systemic immune-inflammation index

over 42% exhibited thyroid dysfunction; 34% had overt hypothyroidism, and 8.7% subclinical hypothyroidism, which reflects the dynamic spectrum of thyroid damage. Euthyroidism in HT may indicate an early or compensatory phase[2] of the disease where the gland is still able to meet hormonal demands despite underlying autoimmunity, whereas progression to overt hypothyroidism suggests a more advanced stage of glandular destruction with potential implications for metabolic and cardiovascular risk.[2,19,23] Euthyroid status was better maintained following surgical interventions such as thyroidectomy, while hormonal treatments were ineffective against the progression of the disease.[24]

The marked elevation of anti-thyroid antibodies, both anti-TG and anti-TPO in the HT group, is consistent with the autoimmune nature of the disease.[25,26] These antibodies not only confirm the diagnosis of HT but may also serve as predictors for the progression of hypothyroidism.[23,27] Although the detection of TPO antibodies in the sera has a predictive value, only 75% of the patients diagnosed with HT suggest circulating autoantibodies are the outcome but not the initiator of the disease.[3,28] The significantly lower TG levels observed in HT patients further support the notion of a destructive process in the thyroid gland, where the loss of functional thyroid tissue corresponds with diminished hormone synthesis.[3,26]

The comparison of inflammatory markers in the circulation, such as the SIRI, SII, NLR, PLR, and others, revealed no significant differences between the HT and control groups. Similarly, Bozdag et al.[9] found that NLR and PLR failed to differentiate neither HT vs control groups nor between euthyroid, hypothyroid, and subclinical hypothyroid patients, whereas hematologic markers like NLR, PLR, and SII were deemed impractical in pediatric HT patients.[11] In contrast, two of the studies provide evidence that NLR[8] and PLR[8,10] were significantly elevated in HT patients, and PLR was able to differentiate euthyroid and hypothyroid patients.[10]

Limitations

The cross-sectional design prevents us from establishing causal relationships between metabolic factors and HT. While we analyzed various inflammatory markers, other potential contributors to immune activation, such as cytokine profiles, were not included. Also, the study did not assess dietary habits, which have been shown to influence thyroid function and inflammation.

CONCLUSION

In conclusion, our study found no significant differences in inflammatory indices between HT patients and euthyroid controls, despite the clear thyroid dysfunction and elevated thyroid autoantibodies, anti-TG, and anti-TPO in the HT group. The gender differences and correlation with anthropometric measures emphasize the complex interplay between metabolic factors and thyroid autoimmunity. Further longitudinal studies incorporating dietary, genetic, and cytokine analyses are needed to elucidate the underlying mechanisms of HT progression and inflammation.

ETHICAL DECLARATIONS

Ethics Committee Approval

Necessary ethical compliance and approvals were granted by the Karabük University Non-interventional Clinical Researches Ethics Committee (Date: 06.01.2025, Decision No: 2025/2055).

Informed Consent

All patients signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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