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Research Article

Thyroid autoantibody status and cardiometabolic characteristics in euthyroid Hashimoto's thyroiditis

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Abstract

Background: Hashimoto's thyroiditis (HT) is commonly characterized by circulating thyroid autoantibodies, although some patients exhibit characteristic ultrasonographic features despite negative antibody status. Whether thyroid autoantibody status is associated with metabolic characteristics in euthyroid HT remains unclear.

Aim: To compare metabolic and cardiometabolic characteristics according to thyroid autoantibody status in untreated euthyroid patients with HT.

Methods: This retrospective, single-center study included 163 untreated euthyroid patients with HT, classified as antibody-positive (Ab+; $n = 71$) or antibody-negative (Ab–; $n = 92$). Anthropometric, biochemical, and metabolic parameters were assessed, including body mass index (BMI), homeostatic model assessment for insulin resistance (HOMA-IR), atherogenic index of plasma (AIP), and triglyceride-glucose (TyG) index. Between-group analyses were adjusted for age, sex, and BMI.

Results: In unadjusted analyses, Ab– patients had higher BMI, fasting insulin, HOMA-IR, triglycerides, AIP, TyG index, and uric acid levels and lower HDL cholesterol levels. After adjustment, differences persisted for triglycerides ($p = 0.005$), HDL cholesterol ($p = 0.044$), AIP ($p = 0.003$), and TyG index ($p = 0.006$), whereas fasting insulin, HOMA-IR, and uric acid were no longer significantly different. Thyroid peroxidase antibody (TPOAb) concentrations showed weak inverse correlations with fasting insulin, HOMA-IR, triglycerides, AIP, and TyG index, whereas thyroglobulin antibody (TgAb) concentrations showed no significant associations with the examined metabolic parameters.

Conclusion: Thyroid autoantibody status was associated with selected cardiometabolic characteristics in untreated euthyroid patients with HT. Although adiposity accounted for part of the initially observed differences, selected lipid-related differences persisted after adjustment for age, sex, and BMI. These findings warrant confirmation in prospective studies.

Keywords

cardiometabolic risk, euthyroidism, Hashimoto's thyroiditis, metabolic profile, thyroid autoantibodies

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PENSOFT

Introduction

Chronic lymphocytic thyroiditis, also known as autoimmune thyroiditis or Hashimoto's thyroiditis (HT), is the most common organ-specific autoimmune disease and the most common cause of thyroid dysfunction in iodine-sufficient areas (Ragusa et al. 2019). The disease affects almost 10% of the population, predominantly women, and leads to progressive thyroid dysfunction as a result of infiltration by inflammatory cells and fibrosis of the thyroid parenchyma.

Both cellular (T-cell-mediated) and humoral (B-cell-mediated) immune responses have roles in the pathogenesis of this disorder (Ragusa et al. 2019). Thyroid antigens can be released due to environmental factors such as infections, excessive iodine intake, or radiation exposure. Dendritic cells and other antigen-presenting cells (APCs) process these antigens and migrate to lymph nodes, where they present antigenic markers to naive T cells. Pro-inflammatory cytokines, including interferon gamma (IFN-γ), interleukin 17 (IL-17), and tumor necrosis factor alpha (TNF-α), are involved in this immune response (Vargas-Uricoechea 2023). B cells also contribute to HT through the production of autoantibodies against the thyroid antigens thyroglobulin and thyroid peroxidase. Thyroglobulin antibodies (TgAb) and thyroid peroxidase antibodies (TPOAb) are present in most patients and serve as useful diagnostic markers (Caturegli et al. 2014). However, the positive predictive value of TPOAb for HT is not absolute (Guan et al. 2019), and some patients have characteristic ultrasonographic findings despite negative circulating antibodies (Guan et al. 2019; Croce et al. 2020).

The immunopathogenesis of antibody-negative HT is thought to involve predominantly cell-mediated immune mechanisms, including natural killer cells, regulatory T-cell dysfunction, local intrathyroidal antibody production, and altered immune regulation influenced by vitamin D deficiency and epigenetic mechanisms (Rydzewska et al. 2018; Ragusa et al. 2019; Vargas-Uricoechea 2023; Bendotti et al. 2026). Although these mechanisms have been proposed, their clinical and metabolic implications remain incompletely understood.

Thyroid hormones affect energy homeostasis, lipid and glucose metabolism, and blood pressure (Delitala et al. 2017). Although thyroid autoantibodies are well-established markers of autoimmune thyroid disease, their relationship with metabolic disturbances in euthyroid patients remains controversial. Some studies have reported associations between thyroid autoantibodies and disturbances in glucose metabolism, lipid metabolism, and cardiometabolic risk profiles (Wu et al. 2020), whereas others have found no significant relationship between TPOAb and lipid profiles (Wells and Hueston 2005). Evidence directly comparing metabolic and cardiometabolic profiles between antibody-positive and antibody-negative euthyroid patients with HT remains limited.

Clarifying these associations may improve the understanding of cardiometabolic risk stratification in euthyroid patients with HT and contribute to more individualized clinical follow-up.

The aim of this study was to compare metabolic and cardiometabolic risk markers in euthyroid patients with HT according to thyroid autoantibody status and to investigate the associations between thyroid autoantibody status and parameters of glucose and lipid metabolism.

Materials and methods

Study design and participants

A total of 163 euthyroid patients diagnosed with HT were included in this retrospective, single-center study. Patients were categorized according to thyroid autoantibody status into an antibody-positive group (Ab+; $n = 71$) and an antibody-negative group (Ab–; $n = 92$). For the Ab+ group, the diagnosis of HT was based on elevated serum thyroid autoantibodies (TPOAb > 9 IU/L or TgAb > 4 IU/L, or both). Patients in the Ab– group had TPOAb and TgAb concentrations below these thresholds and an established diagnosis based on previous clinical evaluation and characteristic ultrasonographic findings, including diffuse hypoechogenicity and heterogeneous thyroid echotexture. Thyroid ultrasonography was performed in all participants as part of the diagnostic evaluation. Patients were recruited from the Department of Endocrinology of St. George University Hospital, Plovdiv, Bulgaria. The study was based on the retrospective analysis of routinely collected clinical data. All patients had signed written informed consent upon hospital admission for the use of their clinical data for research purposes.

Hormonal and biochemical measurements

Fasting plasma glucose (FPG), uric acid (UA), total cholesterol (TC), HDL cholesterol (HDL-C), and triglycerides (TG) were measured using a Konelab 60i clinical chemistry analyzer (Thermo Electron Corporation, Finland). Immunoreactive insulin (IRI), thyroid-stimulating hormone (TSH), free triiodothyronine (FT3), free thyroxine (FT4), TPOAb, and TgAb were determined by electrochemiluminescence immunoassay (ECLIA) using the ACCESS analyzer.

Calculated metabolic indices

BMI, AIP, HOMA-IR, and TyG index were calculated using standard formulas.

Inclusion and exclusion criteria

Inclusion criteria comprised a confirmed diagnosis of HT, clinical and biochemical euthyroidism at the time of assessment, and the absence of levothyroxine replacement therapy. Patients receiving levothyroxine or other medications known to influence thyroid hormone status, carbohydrate or lipid metabolism, or body weight were

excluded from the present analysis. Participants with acute or chronic diseases potentially affecting the evaluated metabolic parameters were also excluded. Participants were questioned regarding iodine supplementation and potential excessive iodine exposure.

Statistical analysis

The distribution of each continuous variable was assessed separately in the two antibody groups using the Shapiro–Wilk test. Variables that were approximately normally distributed in both groups are presented as mean ± standard deviation (SD) and were compared using the independent-samples *t*-test. All other continuous variables are presented as median (25^{th}–75^{th} percentiles) and were compared using the Mann–Whitney *U* test. Categorical variables are presented as *n* (%) and were compared using Pearson's chi-square test. Because BMI was the only baseline characteristic that differed between the groups, the between-group analyses were repeated using analysis of covariance adjusted for age, sex, and BMI. Right-skewed outcomes were log-transformed before analysis, and their group effects are reported as ratios of geometric means. Associations between thyroid autoantibodies and metabolic, lipid, and cardiometabolic markers were assessed using Spearman rank correlations. The significance level was set at $p < 0.05$. Statistical analyses were performed using IBM SPSS Statistics, v. 23.

Results

The Ab+ group included 71 patients (63 women and eight men), whereas the Ab− group comprised 92 patients (75 women and 17 men); the difference in sex distribution was not significant, $\chi^2(1, N = 163) = 1.61$, $p = 0.205$. Basic biochemical and hormonal characteristics of the patients are summarized in Table 1.

No significant differences were observed between the groups regarding age, thyroid function parameters (TSH, FT3, and FT4), total cholesterol, or liver enzyme levels (ASAT and ALAT) (all $p > 0.05$).

In contrast, the Ab− group exhibited a significantly less favorable metabolic profile. Compared with Ab+ patients, they had a higher BMI ($p = 0.029$), fasting insulin ($p = 0.019$), and HOMA-IR ($p = 0.007$), indicating greater insulin resistance. They also demonstrated higher triglyceride levels ($p = 0.006$), AIP ($p = 0.003$), TyG index ($p = 0.007$), and uric acid concentrations ($p = 0.008$), whereas HDL cholesterol levels were significantly lower ($p = 0.038$).

After adjustment for age, sex, and BMI, Ab+ patients had 22% lower triglyceride concentrations than Ab− patients (geometric mean ratio = 0.78, 95% CI [0.65, 0.92], $p = 0.005$). They also had lower AIP (adjusted mean difference = −0.15, 95% CI [−0.24, −0.05], $p = 0.003$) and TyG index values (adjusted mean difference = −0.37, 95% CI [−0.64, −0.11], $p = 0.006$). No adjusted differences were found in fasting insulin, HOMA-IR, or uric acid (Table 2).

Categorical cardiometabolic risk indicators

Each parameter was additionally analyzed against its laboratory reference range or established risk cutoff. Categorical cardiometabolic characteristics were generally similar between the antibody groups (Table 3). However, Ab− patients had a higher prevalence of obesity (54.5% vs. 27.5%), $\chi^2(2) = 14.55$, $p < 0.001$, Cramér's $V = 0.316$. The distribution of AIP risk categories also differed between groups, $\chi^2(2) = 6.49$, $p = 0.039$, Cramér's $V = 0.204$, with high AIP risk being more frequent in Ab− patients (24.7% vs. 10.4%). No other categorical characteristics differed significantly between the groups.

Table 1. Demographic, hormonal, biochemical, and metabolic characteristics of euthyroid patients with HT according to thyroid autoantibody status.

Characteristic	Ab+ (*n* = 71)	Ab− (*n* = 92)	Test statistic	*p*
Age (years)	43.00 [32.50–55.00]	37.50 [31.50–54.00]	$U = 2899.0$, $Z = -1.229$	0.219
BMI (kg/m²)	24.85 [23.34–33.90]	31.22 [24.30–35.08]	$U = 1630.5$, $Z = -2.184$	**0.029**
Fasting glucose (mmol/L)	5.20 [4.82–5.88]	5.31 [4.89–6.03]	$U = 2451.5$, $Z = -0.951$	0.343
Fasting insulin (μIU/mL)	4.92 [3.44–9.78]	7.04 [5.40–11.99]	$U = 1130.0$, $Z = -2.346$	**0.019**
HOMA-IR	1.00 [0.73–2.41]	1.85 [1.17–3.40]	$U = 855.0$, $Z = -2.699$	**0.007**
Total cholesterol (mmol/L)	4.96 [4.25–5.84]	5.10 [4.34–5.95]	$U = 3017.0$, $Z = -0.151$	0.880
HDL-cholesterol (mmol/L)	1.36 [1.20–1.62]	1.26 [1.10–1.53]	$U = 2403.5$, $Z = -2.070$	**0.038**
Triglycerides (mmol/L)	0.89 [0.65–1.17]	1.23 [0.79–1.72]	$U = 2241.5$, $Z = -2.745$	**0.006**
AIP	−0.23 [−0.36–0.02]	0.00 [-0.27–0.21]	$U = 2149.0$, $Z = -2.981$	**0.003**
TyG index	8.21 [7.89–8.65]	8.62 [7.99–9.05]	$U = 1907.0$, $Z = -2.696$	**0.007**
Uric acid (μmol/L)	265.00 [221.50–328.00]	318.00 [245.00–408.00]	$U = 2245.5$, $Z = -2.635$	**0.008**
ASAT (U/L)	19.60 [16.30–24.80]	21.60 [17.40–25.70]	$U = 2626.0$, $Z = -0.975$	0.330
ALAT (U/L)	16.00 [12.40–22.20]	18.50 [12.70–27.30]	$U = 2635.0$, $Z = -0.642$	0.346
TSH (mIU/L)	2.47 [1.81–3.71]	2.31 [1.72–3.53]	$U = 3012.5$, $Z = -0.5025$	0.581
FT3 (pmol/L)	5.50 ± 0.77	5.35 ± 0.78	$t(121) = 1.08$	0.283
FT4 (pmol/L)	11.23 [10.38–12.32]	11.49 [10.05–12.52]	$U = 3020.0$, $Z = -0.258$	0.797

Note. Normally distributed variables are reported as mean ± SD and tested with the independent-samples *t*-test; non-normally distributed variables are reported as median [25^{th}–75^{th} percentiles] and tested with the Mann–Whitney *U* test.

Table 2. Between-group differences in metabolic and cardiometabolic parameters, adjusted for age, sex, and BMI.

Characteristic	Ab+ vs. Ab− [95% CI]	*F*	*p*
Fasting insulin (mIU/L)	0.86 [0.62, 1.19]	$F(1, 92) = 0.89$	0.347
HOMA-IR	0.75 [0.53, 1.04]	$F(1, 83) = 3.05$	0.084
HDL-cholesterol (mmol/L)	1.10 [1.003, 1.200]	$F(1, 123) = 4.15$	0.044
Triglycerides (mmol/L)	0.78 [0.65, 0.92]	$F(1, 124) = 8.19$	0.005
AIP	−0.15 [−0.24, −0.05]	$F(1, 123) = 9.19$	0.003
TyG index	−0.37 [−0.64, −0.11]	$F(1, 115) = 7.72$	0.006
Uric acid (μmol/L)	0.97 [0.89, 1.06]	$F(1, 123) = 0.57$	0.452

Note. All variables are log-transformed and reported as ratios of geometric means, such that a value below 1.00 indicates lower concentrations in Ab+ patients; untransformed variables (AIP and TyG index) are reported as adjusted mean differences (Ab+ minus Ab−). The AIP and TyG indices are logarithmic by construction and were not transformed.

Correlations between thyroid autoantibodies and metabolic parameters

TgAb and TPOAb were moderately positively correlated (Spearman's $\rho = 0.539$, $p < 0.01$). Higher TPOAb concentrations were weakly associated with lower fasting insulin ($\rho = -0.205$, $p < 0.05$) and lower HOMA-IR ($\rho = -0.230$, $p < 0.05$) but were not associated with fasting glucose. TgAb concentrations were not associated with any glucose metabolism marker. As expected, fasting glucose and insulin were positively correlated with HOMA-IR ($\rho = 0.564$ and 0.964, respectively; both $p < 0.01$). All pairwise Spearman correlations are presented in Table 4.

Among the lipid and cardiometabolic parameters, TPOAb correlated negatively with AIP ($\rho = -0.243$, $p = 0.005$), triglycerides ($\rho = -0.221$, $p = 0.010$), and TyG index ($\rho = -0.209$, $p = 0.020$) and showed no correlation with total cholesterol, HDL cholesterol, uric acid, or BMI (Table 5). TgAb did not correlate with any lipid or cardiometabolic parameter.

Discussion

The present study compared metabolic and cardiometabolic characteristics of untreated euthyroid patients with HT according to thyroid autoantibody status. In the unadjusted analyses, Ab− patients exhibited a generally less favorable metabolic profile, characterized by higher BMI, fasting insulin, HOMA-IR, triglycerides, AIP, TyG index, and uric acid, together with lower HDL cholesterol. Importantly, adjustment for age, sex, and BMI substantially refined these findings. Differences in fasting insulin, HOMA-IR, and uric acid were attenuated and were no longer statistically significant, whereas differences in triglycerides, HDL cholesterol, AIP, and TyG index persisted. These findings indicate that adiposity may account for part of the initially observed metabolic differences between the antibody groups, while selected lipid-related and cardiometabolic characteristics remained significantly different after adjustment for age, sex, and BMI.

Table 3. Categorical cardiometabolic and clinical characteristics by antibody status.

Characteristic	Ab+ *n* (%)	Ab− *n* (%)	Test statistic	*p*	Effect size
BMI category			**$\chi^2(2) = 14.55$**	**< 0.001**	***V* = 0.316**
Normal	43 (62.3)	24 (31.2)			
Obesity (I-IV)	19 (27.5)	42 (54.5)			
Overweight	7 (10.1)	11 (14.3)			
AIP risk category			**$\chi^2(2) = 6.49$**	**0.039**	***V* = 0.204**
Low	55 (82.1)	57 (64.0)			
Intermediate	5 (7.5)	10 (11.2)			
High	7 (10.4)	22 (24.7)			
HOMA-IR			**$\chi^2(1) = 2.49$**	**0.115**	**OR = 0.43 [0.17, 1.09]**
No	33 (80.5)	39 (63.9)			
Yes	8 (19.5)	22 (36.1)			
Fasting glucose within reference range			**$\chi^2(1) = 0.00$**	**1.000**	**OR = 1.04 [0.49, 2.20]**
No	16 (24.6)	21 (25.3)			
Yes	49 (75.4)	62 (74.7)			
HDL-C			**$\chi^2(2) = 3.25$**	**0.197**	***V* = 0.144**
Below	7 (10.4)	17 (19.1)			
Normal	42 (62.7)	56 (62.9)			
Above	18 (26.9)	16 (18.0)			
Triglycerides			**$\chi^2(2) = 1.31$**	**0.519**	***V* = 0.091**
Below	2 (3.0)	1 (1.1)			
Normal	51 (76.1)	65 (72.2)			
Above	14 (20.9)	24 (26.7)			
Total cholesterol			**$\chi^2(2) = 0.23$**	**0.893**	***V* = 0.038**
Below	1 (1.5)	2 (2.2)			
Normal	33 (48.5)	41 (45.6)			
Above	34 (50.0)	47 (52.2)			

Note. $N = 163$. Percentages are column percentages within each antibody group, based on valid cases. Odds ratios are reported for 2 × 2 tables and Cramér's *V* for larger tables.

Table 4. Spearman correlations between thyroid autoantibodies and parameters of glucose metabolism, $N = 163$.

	TgAb	TPOAb	Fasting glucose	Fasting insulin	HOMA-IR
TgAb	—	0.539**	0.100	0.126	0.116
TPOAb		—	−0.044	−0.205*	−0.230*
Fasting glucose			—	0.400**	0.564**
Fasting insulin				—	0.964**
HOMA-IR					—

Note. * $p < 0.05$. ** $p < 0.01$.

The attenuation of the differences in fasting insulin and HOMA-IR after adjustment for BMI is particularly relevant given the well-established relationship between adiposity, chronic low-grade inflammation, and insulin resistance. Adipose tissue is increasingly recognized as an active immunometabolic organ, and obesity-related inflammation contributes to impaired insulin signaling and broader metabolic dysfunction (Hotamisligil 2017). In addition, thyroid dysfunction, adiposity, insulin resistance, and other components of metabolic syndrome are closely interconnected, making BMI an important potential confounder when metabolic characteristics are compared according to thyroid autoantibody status (Mehran et al. 2019). The persistence of differences in triglycerides, HDL cholesterol, AIP, and TyG index after adjustment suggests, however, that the observed variation in selected cardiometabolic parameters cannot be attributed solely to differences in BMI.

The categorical analyses provided additional clinical context for these findings. Obesity was substantially more frequent among Ab− patients, further emphasizing the importance of adiposity in the interpretation of the unadjusted metabolic differences. High-risk AIP values were also more frequent in antibody-negative individuals, whereas categorical differences in HOMA-IR, fasting glucose, HDL cholesterol, triglycerides, and total cholesterol were not statistically significant. Taken together with the adjusted continuous-variable analyses, these results suggest that the association with thyroid autoantibody status may be more evident for selected lipid-related cardiometabolic measures than for metabolic risk as a whole.

The correlation analysis provided complementary, although modest, evidence regarding the relationship between thyroid autoantibodies and metabolic parameters. Higher TPOAb concentrations were weakly associated with lower fasting insulin and HOMA-IR and were also weakly inversely correlated with triglycerides, AIP, and TyG index. In contrast, TgAb concentrations were not significantly associated with the examined glucose, lipid, or cardiometabolic parameters. The magnitude of the observed TPOAb correlations was small, and these associations should therefore be interpreted cautiously. In particular, they should not be considered evidence of a direct metabolic effect or a protective role of thyroid autoantibodies.

Previous studies examining the relationship between thyroid autoimmunity and metabolic risk in euthyroid populations have yielded inconsistent results. Thyroid autoimmunity in the absence of overt or subclinical hypothyroidism does not necessarily appear to be accompanied by an adverse metabolic profile, although the available evidence remains heterogeneous (Duntas and Brenta 2012; Rotondi et al. 2014). Zhang et al. (2020), in a national survey, reported that TgAb positivity in euthyroid individuals was associated with lower risks of selected glucose and lipid metabolic abnormalities, including favorable associations with triglycerides and HDL-C. Similarly, Raposo et al. (2019) found an inverse association between TPOAb positivity and metabolic syndrome, particularly hypertriglyceridemia. More recently, a Mendelian randomization study identified an inverse association between genetic susceptibility to TPOAb positivity and metabolic syndrome, including its triglyceride component (Chen et al. 2024). Although these findings are broadly compatible with the persistence of triglyceride-related differences observed in the present study, neither previous evidence nor the present results support interpreting thyroid autoimmunity itself as metabolically protective.

Conversely, other studies have reported neutral or less favorable metabolic associations with thyroid autoimmunity. Kim et al. (2021), in a nationwide population-based study of euthyroid individuals, reported a higher prevalence of metabolic syndrome and selected cardiometabolic abnormalities, including abdominal obesity and low HDL-C, among TPOAb-positive individuals. Amouzegar et al. (2015), however, found no significant difference in insulin resistance according to thyroid antibody status in euthyroid individuals. Such apparently conflicting findings emphasize the complexity of the relationship between

Table 5. Spearman correlations between thyroid autoantibodies and lipid and cardiometabolic parameters, $N = 163$.

	TgAb	TPOAb	Total cholesterol	Triglycerides	HDL-cholesterol (mmol/L)	TyG	AIP	Uric acid (μmol/L)	BMI
TgAb	—	0.539**	0.014	0.024	0.068	0.084	0.018	0.020	−0.009
TPOAb		—	−0.066	−0.221*	0.155	−0.209*	−0.243**	−0.024	−0.162
Total cholesterol			—	0.567**	0.280**	0.585**	0.366**	0.085	0.341**
Triglycerides				—	−0.330**	0.970**	0.943**	0.391**	0.456**
HDL-cholesterol (mmol/L)					—	−0.342**	−0.601**	−0.316**	−0.240**
TyG						—	0.918**	0.347**	0.450**
AIP							—	0.428**	0.479**
Uric acid (μmol/L)								—	0.467**
BMI									—

Note. * $p < 0.05$. ** $p < 0.01$.

thyroid autoimmunity and metabolic health. Differences in study populations, adiposity, thyroid function within the reference range, definitions of antibody positivity, and statistical adjustment for potential confounders may all contribute to the heterogeneity of published results.

The biological mechanisms underlying these associations remain incompletely understood and are likely to involve complex interactions between immune activation, adipose tissue inflammation, insulin signaling, and lipid metabolism. Immune–metabolic cross-talk has increasingly been recognized as an important link between chronic low-grade inflammation and metabolic dysfunction (Hotamisligil 2017). However, the present findings do not allow determination of whether thyroid autoantibodies themselves contribute to metabolic variation or instead represent markers of an underlying immunometabolic phenotype. The cross-sectional nature of the study also precludes establishing the temporal direction of these relationships.

From a clinical perspective, the persistence of differences in triglycerides, HDL cholesterol, AIP, and TyG index after adjustment for age, sex, and BMI suggests that thyroid autoantibody status may be associated with selected aspects of cardiometabolic risk beyond differences in adiposity alone. Conversely, the loss of statistical significance for fasting insulin and HOMA-IR after adjustment indicates that the initially observed differences in insulin resistance were substantially influenced by BMI. Accordingly, the present findings do not support the use of thyroid autoantibody status as an independent marker of overall metabolic risk. Rather, they identify an association with selected cardiometabolic characteristics that warrants further investigation and confirmation in prospective studies. From a practical standpoint, metabolic risk assessment in patients with HT should therefore continue to be based primarily on established metabolic risk factors, while antibody status may represent an additional characteristic deserving further study.

The strengths of the present study include the assessment of a well-defined cohort of untreated euthyroid patients with HT, the direct comparison of Ab+ and Ab− groups, and the comprehensive evaluation of glucose, lipid, and cardiometabolic parameters, including HOMA-IR, TyG index, and AIP. Restricting the analysis to untreated euthyroid patients reduced potential confounding related to thyroid hormone replacement therapy. Importantly, the adjusted analyses accounted for age, sex, and BMI, allowing the contribution of adiposity to the observed between-group differences to be evaluated more appropriately. The combined analysis of continuous metabolic variables, categorical risk indicators, and antibody titers also provided complementary perspectives on the observed associations.

The present study also has several limitations. Its retrospective cross-sectional design precludes causal inference, and the single-center setting may limit the generalizability of the findings. Although the principal analyses were adjusted for age, sex, and BMI, residual confounding related to adiposity and other unmeasured metabolic factors cannot be excluded. BMI does not fully reflect body composition or visceral adiposity, and information on waist circumference, dietary habits, physical activity, smoking status, and disease duration was not consistently available. Furthermore, the correlations between thyroid autoantibody concentrations and metabolic parameters were generally weak and should be considered exploratory. Prospective longitudinal studies incorporating more detailed measures of body composition, lifestyle factors, and disease duration are needed to clarify the relationship between thyroid autoantibody status and cardiometabolic characteristics in euthyroid HT.

Conclusion

In conclusion, euthyroid patients with HT showed differences in selected metabolic and cardiometabolic characteristics according to thyroid autoantibody status. Although Ab− patients exhibited a generally less favorable metabolic profile in the unadjusted analyses, adjustment for age, sex, and BMI attenuated several of these differences. Significant differences persisted for triglycerides, HDL cholesterol, AIP, and TyG index, whereas differences in fasting insulin, HOMA-IR, and uric acid were no longer statistically significant. These findings suggest that adiposity contributes to some of the observed metabolic differences, while selected lipid-related cardiometabolic characteristics remained significantly different between the antibody groups after adjustment for age, sex, and BMI. Given the retrospective cross-sectional design, these associations should be interpreted cautiously and do not establish a causal or protective role of thyroid autoimmunity. Further prospective studies are needed to confirm these findings and determine their clinical significance.

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Additional information

Conflict of interest

The authors have declared that no competing interests exist.

Ethical statements

The authors declared that no clinical trials were used in the present study.

The authors declared that no experiments on humans or human tissues were performed for the present study.

All patients had signed written informed consent upon hospital admission for the use of their clinical data for research purposes.

The authors declared that no experiments on animals were performed for the present study.

The authors declared that no commercially available immortalised human and animal cell lines were used in the present study.

Artificial Intelligence (AI) use

The authors accept full responsibility for the content of the manuscript, including the disclosure of any use of AI.
No AI tools were used in the preparation of this manuscript.

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Author contributions

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Data availability

All of the data that support the findings of this study are available in the main text.