RESEARCH ARTICLE / ARAŞTIRMA MAKALESİ

DOI: 10.4274/atfm.galenos.2025.54036
J Ankara Univ Fac Med 2025;78(1):42-48

MEDICAL SCIENCES / DAHİLİ TIP BİLİMLERİ

Evaluation of Triglyceride-Glucose Index in Patients with Hashimoto's Thyroiditis

Hashimoto Tiroiditli Hastalarda Trigliserit-Glukoz İndeksinin Değerlendirilmesi

Oğuzhan Zengin[1], Burak Göre[2], Mustafa Doğru[3], Öztuğ Aytekin[4], Elif Ünal[5], Oğuz Öztürk[6], Enes Seyda Şahiner[1], Osman İnan[1], İhsan Ateş[1]

[1]Ankara Bilkent City Hospital, Clinic of Internal Medicine, Ankara, Türkiye
[2]Çerkeş State Hospital, Clinic of Internal Medicine, Çankırı, Türkiye
[3]Gaziantep City Hospital, Clinic of Internal Medicine, Gaziantep, Türkiye
[4]Bozüyük State Hospital, Clinic of Internal Medicine, Bilecik, Türkiye
[5]Esenyurt Necmi Kadıoğlu State Hospital, Clinic of Internal Medicine, İstanbul, Türkiye
[6]Ankara Bilkent City Hospital, Clinic of Gastroenterology, Ankara, Türkiye

Abstract

Objectives: Recently, researchers have used the triglyceride-glucose (TyG) index for the possibility of metabolic syndrome and insulin resistance. Although altered glucose and lipid metabolism have been observed in patients with Hashimoto's thyroiditis (HT), the impact of autoimmune factors on the TyG index in euthyroid HT patients has not been sufficiently studied. This research sought to assess the effects of autoimmunity on the TyG index in euthyroid HT patients.

Materials and Methods: Our research was planned as a retrospective, single-center, observational study. The study included 50 participants diagnosed with HT and 52 control groups without thyroid disease, who were followed up in the internal medicine service and outpatient clinic.

Results: The patient group had a mean age of 44.54±13.72 years, while the control group had a mean age of 33.5±13.17 years. The HT group was significantly older ($p<0.001$). No meaningful distinctions were detected regarding the gender distribution between the groups ($p=0.357$). In the HT group, the low-density lipoprotein (LDL) cholesterol level was 137.8±41.5 mg/dL, and the non-high-density lipoprotein (HDL) cholesterol level was 160.5±45.2 mg/dL, both of which were significantly higher compared to the control group (LDL: $p=0.046$, non-HDL: $p=0.029$). The TyG index showed similar results between the two groups, with measurements of 8.6±1.2 in the HT group and 8.4±1.3 in the control group ($p=0.121$).

Conclusion: In contrast to previous studies, our study focused on the impact of autoimmunity on TyG index changes in patients with euthyroid HT, taking into account the relationship with thyroid hormones. Accordingly, a similar correlation was detected between the TyG index and age in patients with and without HT. As a result, we found that the TyG index did not change due to autoimmunity in patients with euthyroid HT.

Keywords: Triglyceride/glucose index, Hashimoto's thyroiditis, autoimmunity, marker, thyroid hormone

Öz

Amaç: Son zamanlarda araştırmacılar, metabolik sendrom ve insülin direnci olasılığını tespit etmek için bir tarama yöntemi olarak trigliserit-glukoz (TyG) indeksini kullanmaya başladı. Hashimoto tiroiditi (HT) hastalarında glukoz ve lipit metabolizmasında değişiklik gözlenmesine rağmen, ötiroid HT hastalarında otoimmün faktörlerin TyG indeksi üzerindeki etkisi yeterince araştırılmamıştır. Bu araştırma, ötiroid HT hastalarında otoimmünitenin TyG indeksi üzerindeki etkilerini değerlendirmeyi amaçladı.

Gereç ve Yöntem: Araştırmamız retrospektif, tek merkezli, gözlemsel bir çalışma olarak planlandı. Çalışmaya dahiliye servisinde ve polikliniğinde takip edilen HT tanısı alan 50 katılımcı ve tiroid hastalığı olmayan 52 kontrol grubu dahil edildi.

Address for Correspondence/Yazışma Adresi: Burak Göre, MD
Çerkeş State Hospital, Clinic of Internal Medicine, Çankırı, Türkiye
E-mail: goreburak1@gmail.com **ORCID ID:** orcid.org/0000-0002-6082-8323

Received/Geliş Tarihi: 22.11.2024 **Accepted/Kabul Tarihi:** 06.02.2025 **Publication Date/Yayınlanma Tarihi:** 21.03.2025

Cite this article as/Atıf: Zengin O, Göre B, Doğru M, et al. Evaluation of triglyceride-glucose index in patients with Hashimoto's thyroiditis. J Ankara Univ Fac Med. 2025;78(1):42-48

Bulgular: Hasta grubunun yaş ortalaması 44,54±13,72 yıl, kontrol grubunun yaş ortalaması ise 33,5±13,17 yıl idi. HT grubu anlamlı derecede daha yaşlıydı (p<0,001). Gruplar arasında cinsiyet dağılımı açısından anlamlı bir farklılık saptanmadı (p=0,357). HT grubunda düşük yoğunluklu lipoprotein (LDL) kolesterol düzeyi 137,8±41,5 mg/dL ve yüksek yoğunluklu lipoprotein (HDL) olmayan kolesterol düzeyi 160,5±45,2 mg/dL olup her ikisi de kontrol grubuna göre anlamlı derecede yüksekti (LDL: p=0,046, HDL olmayan: p=0,029). TyG indeksi, HT grubunda 8,6±1,2 ve kontrol grubunda 8,4±1,3 ölçümleriyle iki grup arasında benzer sonuçlar gösterdi (p=0,121).

Sonuç: Çalışmamızda önceki çalışmalardan farklı olarak ötiroid HT hastalarında tiroid hormonları ile ilişkisi dikkate alınarak otoimmünitenin TyG indeksi değişiklikleri üzerindeki etkisine odaklanılmıştır. Buna göre HT olan ve olmayan hastalarda TyG indeksi ile yaş arasında benzer bir korelasyon tespit edildi. Sonuç olarak ötiroid HT hastalarında TyG indeksinin otoimmüniteye bağlı olarak değişmediğini tespit ettik.

Anahtar Kelimeler: Trigliserit/glukoz indeksi, Hashimoto tiroiditi, otoimmünite, marker, tiroid hormonu

Introduction

Thyroid dysfunction may contribute to abnormal glucose homeostasis, as thyroid hormones play an important role in regulating multiple metabolic processes. Overproduction of thyroid hormones can have an impact on insulin signaling and turnover of glucose metabolism. The reduction in muscle mass among the elderly impacts peripheral glucose uptake, resulting in hyperinsulinemia and insulin resistance (1-3). Both subclinical and overt hypothyroidism have been linked to metabolic syndrome (4-11). A connection between hypothyroidism and metabolic disorders has also been revealed in the literature (12,13). Increases in thyroid stimulating hormone (TSH) levels, even at low levels, have been shown to be associated with insulin resistance (14).

Triglyceride/glucose (TyG) index determined by fasting triglyceride and glucose levels, has recently become a new indicator for metabolic syndrome (15-17). Several studies have found a correlation between elevated TyG levels and metabolic disorders (18-20).

Studies have been conducted in the literature to demonstrate the relationship between HT and dyslipidemia, and it has been found that the prevalence of dyslipidemia is increased in patients with HT (21). Moreover, in hypertension (HTN), cytokines like interleukin-1 beta (IL-1β), IL-6, and tumor necrosis factor alpha are elevated, triggering metabolic processes (22). Research has found that conditions like rheumatoid arthritis and systemic lupus erythematosus, which are autoimmune in nature, are associated with insulin resistance, largely due to inflammatory processes and an elevated TyG index (23).

The literature has not yet studied the relationship between euthyroid HT and TyG. In this study, we wanted to determine the relationship between HT and the TyG index, which is a marker of metabolic disease, in euthyroid patients. We also examined the relationship between TyG and autoimmunity in our study.

Materials and Methods

Our research was planned as a retrospective, single-center, observational study. Patients with a diagnosis of HT who were followed up in the internal medicine service and outpatient clinic with TSH (reference: 0,55-4,78 mU/L), free triiodothyronine (T3) (reference: 2,3-4,2 ng/L) and free levothyroxine (T4) (reference: 0,89-1,76 ng/dL) levels within the normal range and a control group without thyroid disease were included in the study. For the diagnosis of Hashimoto's thyroiditis (HT), antibody levels (anti-thyroid peroxidase) were checked and thyroid ultrasound was performed. The study involved patients who had high antibody levels and ultrasound findings compatible with HT. All actions in this research received approval from the Ankara Bilkent City Hospital Clinical Research Ethics Committee and were executed in line with the ethical principles established in the Declaration of Helsinki and its subsequent updates (date: 12/07/2023, approval no: E2-23-4142). The study was planned in a retrospective manner; as a result, patient consent was not collected.

The number of participants in each group was set at 45 (±10%) using the G*Power software, based on an analysis with a 95% confidence level (1-α), a 95% test power (1-β), and an effect size (d) of 0.5.

The study included 50 patients diagnosed with HT disease and 52 patients in the control group without thyroid disease, aged 18 and over, who were followed up in internal medicine services and outpatient clinics between September 2021 and March 2023. The criteria for exclusion in our study were specified as follows: age under 18, known liver disease, diabetes, HTN, chronic kidney disease, use of lipid-lowering therapy, sepsis, and abnormal thyroid function tests (TSH, T3, T4). Among the participants diagnosed with HT, there were equal numbers of those who received levothyroxine sodium treatment and those who did not. All euthyroid patients in the HT group were included in the study, regardless of whether they received treatment or not.

Various factors were assessed, including the patients' age, gender, existing comorbidities, prescribed medications, complete blood count, levels of white blood cells, neutrophils, lymphocytes, neutrophil-to-lymphocyte ratio, eosinophils, triglycerides, glucose, and lipid profile, along with TSH, T3, and T4.

Statistical Analysis

The data were analyzed utilizing SPSS software, version 25.0 (IBM Corp., 2017). The distribution of variables was checked with histogram graphs and the Kolmogorov-Smirnov test. Descriptive statistics, including mean, standard deviation, and range (minimum-maximum values), were presented. The relationship between laboratory and demographic parameters was assessed using Pearson's correlation coefficient. In patients diagnosed with HT, categorical variables such as comorbidities, demographic data, lab results, and age were compared using the chi-square test. A p-value below 0.05 was regarded as statistically meaningful.

Results

In this study, laboratory values and comorbidities were compared between patients and the control group. The results indicated that the mean age of the patient group was higher, but no considerable variations were observed in parameters such as sex, neutrophil count, and lymphocyte count. Low-density lipoprotein (LDL), non-high-density lipoprotein (HDL) cholesterol, and the TyG index was notably higher in the patient group. Significant correlations were observed between the TyG index and age, lymphocyte count, HDL, non-HDL cholesterol, and triglyceride levels. There were no cases of diabetes or coronary artery disease in either group, and the rate of HTN was alike in both groups. To conclude, while several laboratory variables were notably different across the groups, there were no substantial variations in comorbidities.

Patients with HT disease and the control group were compared according to the measured parameters in Table 1. The mean age of the patient group was higher ($p<0.001$). The proportion of females in the patient group (72%) was higher than in the control group (63.46%), but there was no statistical significance ($p=0.357$). No notable differences were observed in neutrophil and lymphocyte counts ($p=0.217$ and $p=0.164$, respectively). The neutrophil/lymphocyte ratio did not exhibit a significant difference across the groups ($p=0.079$). The hemoglobin concentrations were similar across both groups, with no considerable variation ($p=0.830$). LDL concentrations were notably elevated in the HT group ($p=0.046$). No notable difference was observed in HDL levels ($p=0.202$). The levels of non-HDL cholesterol were markedly increased in the HT group ($p=0.029$). Triglyceride and glucose levels did not show significant differences ($p=0.213$ and $p=0.543$, respectively). No meaningful variation was detected in the TyG index across the two groups ($p=0.121$).

The relationship of the TyG index with other parameters was compared between the groups in Table 2. When evaluating the relationship between the TyG index and various demographic and laboratory values in Hashimato's patients and the control group, a strong association between age and the TyG index was noted in both groups (Hashimoto: $p=0.009$, $r=0.364$; control: $p=0.003$, $r=0.407$). No notable association was detected between neutrophil count and the TyG index in either group (Hashimoto: $p=0.779$, $r=0.041$; control: $p=0.787$, $r=-0.038$). A positive correlation was found between lymphocyte count and the TyG index in the HT group ($p=0.002$, $r=0.433$), whereas no significant correlation was observed in the control group ($p=0.268$), $r=-0.156$). A negative correlation was observed between the neutrophil/lymphocyte ratio and the TyG index in the HT group ($p=0.015$, $r=-0.342$), but no meaningful correlation

Table 1: Comparison of laboratory values between patient and control groups

		Control	HT	Overall	p-value
		Mean ± SD	Mean ± SD	Mean ± SD	
Age		33.5±13.17	44.54±13.72	38.91±14.48	<0.001[1]
Sex, n (%)	Female	33 (63.46)	36 (72.00)	69 (67.65)	0.357[2]
	Male	19 (36.54)	14 (28.00)	33 (32.35)	
Neutrophil (x10^6/L)		4154.29±1516.07	3786±1474.93	3973.75±1500.08	0.217[1]
Lymphocyte (x10^6/L)		2047.5±543.88	2230.2±751.69	2137.06±657.2	0.164[1]
Neutrophil/lymphocyte ratio		2.33±1.92	1.81±0.73	2.08±1.48	0.079[1]
Hemoglobin (g/dL)		13.6±1.87	13.53±1.38	13.56±1.64	0.830[1]
Low-density lipoprotein (mg/dL)		101.35±28.73	116.16±43.24	108.61±37.14	0.046[1]
High-density lipoprotein (mg/dL)		50.77±15.9	54.52±13.46	52.61±14.8	0.202[1]
Non-high-density lipoprotein (mg/dL)		120.02±31.57	137.38±45.84	128.53±39.98	0.029[1]
Triglyceride (mg/dL)		95.98±51.2	108.36±48.33	102.05±49.96	0.213[1]
Glucose (mg/dL)		87.44±9.97	88.54±8.07	87.98±9.06	0.543[1]
Triglyceride/glucose index		8.23±0.47	8.38±0.48	8.31±0.48	0.121[1]

[1]Independent t-test [2]Chi-square test
p: variation between laboratory parameters with and without Hashimoto, HT: Hashimoto thyroiditis, SD: Standart devitation

was identified in the control group (p=0.238, r=0.167). No significant correlation was found between hemoglobin levels and the TyG index in either group (Hashimoto: p=0.235, r=0.171; control: p=0.463, r=-0.104). A borderline positive correlation was found between LDL and the TyG index in the HT group (p=0.058, r=0.270), but no significant relationship was observed in the control group (p=0.129, r=0.213). A significant negative correlation was found between HDL and the TyG index in both groups (Hashimoto: p=0.002, r=-0.433; control: p<0.001, r=-0.476). A significant positive correlation was observed between non-HDL cholesterol and the TyG index in both groups (Hashimoto: p=0.001, r=0.448; control: p<0.001 r=0.562). A strong positive correlation was found between triglycerides and TyG index in both groups (Hashimoto: p<0.001, r=0.954; control: p<0.001, r=0.916). A positive correlation was also observed between glucose levels and the TyG index in both groups (Hashimoto: p<0.001, r=0.573; control: p=0.002, r=0.417).

Comparison of comorbidities between patient and control groups was shown in Table 3. When comparing comorbid diseases between HT patients and the control group, no cases of diabetes mellitus (DM) were observed in either group. HTN was present in 3.85% of the control group and 6.00% of the HT group. However, this discrepancy was not found to be statistically significant (p=0.675), indicating that HTN prevalence was similar between the two groups. No cases of coronary artery disease were found in either group. In conclusion, there were no significant differences in the prevalence of HTN or coronary artery disease between the two groups, and no cases of DM or coronary artery disease were observed in either group.

Discussion

Even though studies in the literature suggest a link between the TyG index and thyroid function tests, there is a paucity of previous research on this relationship in patients with euthyroid HT. In our study, we aimed to investigate the potential impact of autoimmunity on the TyG in patients with euthyroid hypothyroidism, excluding thyroid function tests. In our study, we did not find a relationship between TyG index and HT. We found that there was no relationship between thyroid autoimmunity and TyG index in patients with normal thyroid function tests.

There are studies in the literature stating that the TyG index varies in DM, coronary artery disease and chronic kidney disease.

Table 2: Comparison of triglyceride/glucose index with demographic and laboratory values in patient and control groups

Triglyceride/glucose index				
	HT		Control	
	r	p-value	r	p-value
Age	0.364	0.009	0.407	0.003
Neutrophil	0.041	0.779	-0.038	0.787
Lymphocyte	0.433	0.002	-0.156	0.268
Neutrophil/lymphocyte ratio	-0.342	0.015	0.167	0.238
Hemoglobin	0.171	0.235	-0.104	0.463
Low-density lipoprotein	0.270	0.058	0.213	0.129
High-density lipoprotein	-0.433	0.002	-0.476	<0.001
Non-high-density lipoprotein	0.448	0.001	0.562	<0.001
Triglyceride	0.954	<0.001	0.916	<0.001
Glucose	0.573	<0.001	0.417	0.002

Pearson correlation test
r: Sample correlation coefficient, p: Population correlation coefficient, HT: Hashimoto thyroiditis

Table 3: Comparison of comorbid diseases between patient and control groups

		Control (n=52)	HT (n=50)	Overall (n=102)	p-value[2]
Diabetes mellitus	Absent	52 (100.00)	50 (100.00)	102 (100.00)	
Hypertension	Absent	50 (96.15)	47 (94.00)	97 (95.10)	0.675[2]
Coronary artery disease	Absent	52 (100.00)	50 (100.00)	102 (100.00)	

[2]Chi-square test
SD: Standard deviation, HT: Hashimoto thyroiditis

(8-10). Therefore, unlike other studies in the literature with such chronic diseases, we did not include these patients in our study.

Choi et al. (24) found a relationship between the TyG and TSH intervals in euthyroid patients. In our study, unlike the one conducted by Choi et al. (24), euthyroid patients were not further categorized due to the limited number of participants and the subjective determination of TSH interval values. Therefore, this relationship was not detected in our study since we did not make additional groupings. In contrast to this study, our patient population was younger, and we minimized the impact on the TyG by excluding chronic diseases.

In the literature, there is no TyG study that directly examines the relationship of euthyroid patients with autoimmune thyroiditis with the control group. Lei et al. (25) evaluated patients with autoimmune thyroid disease as predisposed to lipid metabolism disorders, comparing them with the control group. Lipid metabolism and inflammatory cytokines are thought to contribute to the pathogenesis and progression of autoimmune thyroiditis. We thought that the TyG might change by affecting lipid metabolism.

It was observed that the TyG index measured in the elderly population was closely linked to the risk of frailty (26). Loss of muscle mass in the elderly affects peripheral glucose absorption through decreased muscle mass, leading to a state of hyperinsulinemia and insulin resistance (27). In our study, we found that age and TyG index were associated independently of HT.

Our study found similar fasting blood glucose and lipid profiles in both groups. A stronger correlation between serum thyroid hormone levels and blood lipid metabolism, insulin resistance and inflammatory factors has been observed in the literature (25). In our study, unlike the literature, we examined the change in the TyG index due to autoimmunity rather than the thyroid hormone relationship by taking patients with euthyroid HT.

Similar to our study, a retrospective study by Akyüzlü and Mutlu (28) evaluated 1,280 patients and concluded that the TyG, independent of thyroid hormones, cannot serve as a marker of insulin resistance in individuals with thyroid disease. However, in this study, unlike ours, all thyroid diseases except HT constituted the patient group. We defined the normal TSH interval in our study as 0.55-4.78, while Akyüzlü and Mutlu (28) defined it as 0.5-10.

Studies examining patients with systemic autoimmune inflammatory diseases have suggested the presence of an ongoing inflammatory process (29,30). It has been demonstrated that these systemic autoimmune inflammatory diseases may contribute to the development of other conditions, including metabolic syndrome, HTN, dyslipidemia, and diabetes (31). We examined the relationship between the TyG index, which has been previously shown to be associated with metabolic syndrome, and HT, which is an autoimmune disease, and determined that this index cannot be used in HT. We thought that this result, which was different from the literature, might be related to the milder course of euthyroid HT disease compared to other systemic inflammatory diseases. The literature indicates that anti-thyroid peroxidase (TPO) antibody levels are associated with higher insulin levels in patients with a body mass index (BMI) similar to those with levels exceeding 1000 IU/mL. In the same study, consistent with our findings, no notable variation was observed among the participants with and without HT regarding glucose and lipid concentrations (32). In the research by Biyikli et al. (33), fasting glucose and triglyceride levels were notably elevated in the hypertensive group; however, the TyG index was not assessed. We believe that this discrepancy may be attributed to the exclusion criteria in our study, which accounted for additional factors that could influence fasting glucose and triglyceride levels. In addition, we evaluated that the systemic effect of HT is mostly through thyroid hormones, and in our study, this effect was minimized by including euthyroid patients. There is a need for prospective studies examining the relationship between other early diagnosed systemic autoimmune diseases and the TyG index in patients under treatment.

Study Limitations

The limitations of our study include the age variation between the groups and the inability to assess the deficiency of anti-TPO levels due to the small sample size. We recommend conducting studies examining its relationship with anti-TPO level. We suggest that patients undergo long-term follow-up, and recommend planning studies that explore the relationship between the duration of HT disease. We think that this limitation will be eliminated by prospectively recruiting a large number of patients. Another limitation of our study is the age discrepancy between the groups, as glucose levels were influenced by lipid profile changes associated with age.

We recommend conducting additional studies on autoimmune diseases with a large patient population, after excluding the parameters affecting the TyG in the literature. Because the TyG is cheap and easy, it still maintains its popularity in metabolic syndrome. The TyG index has the potential to be an important screening tool for managing these diseases. Therefore, there is a significant need for more extensive and long-term studies examining the relationship between autoimmune diseases, particularly conditions like HT, and the TyG index. Autoimmune diseases can lead to various metabolic changes due to the interaction between genetic and environmental factors, making the management of such conditions more complex. These types of studies will enhance our understanding of the TyG index's effectiveness as a marker for assessing metabolic risk factors

in autoimmune diseases and provide valuable insights for clinical practice. Moreover, the ability of the TyG index to detect metabolic syndrome and related complications early on offers an important advantage in improving patient management and preventing complications.

Conclusion

In conclusion, no changes in the TyG index were observed in euthyroid Hashimoto's patients due to autoimmunity. However, in future large-scale prospective studies, the relationship between the TyG index and thyroid autoimmune diseases may be investigated in greater detail.

Ethics

Ethics Committee Approval: All actions in this research received approval from the Ankara Bilkent City Hospital Clinical Research Ethics Committee (date: 12/07/2023, approval no: E2-23-4142) and were executed in line with the ethical principles established in the Declaration of Helsinki and its subsequent updates.

Informed Consent: Consent was not obtained since it was a retrospective study.

Footnotes

Authorship Contributions

Surgical and Medical Practices: O.Z., B.G., M.D., Ö.A., E.Ü., O.Ü., E.S.Ş., O.İ., İ.A., Concept: O.Z., B.G., M.D., Ö.A., E.Ü., O.Ü., E.S.Ş., O.İ., İ.A., Design: O.Z., B.G., M.D., Ö.A., E.Ü., O.Ü., E.S.Ş., O.İ., İ.A., Data Collection and/or Processing: O.Z., B.G., M.D., Ö.A., E.Ü., O.Ü., E.S.Ş., O.İ., İ.A., Analysis and/or Interpretation: O.Z., B.G., M.D., Ö.A., E.Ü., O.Ü., E.S.Ş., O.İ., İ.A., Literature Search: O.Z., B.G., M.D., Ö.A., E.Ü., O.Ü., E.S.Ş., O.İ., İ.A., Writing: O.Z., B.G., M.D., Ö.A., E.Ü., O.Ü., E.S.Ş., O.İ., İ.A.

Conflict of Interest: The authors have no conflicts of interest to declare.

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

References

1. Martinez B, Ortiz RM. Thyroid hormone regulation and insulin resistance: insights from animals naturally adapted to fasting. Physiology (Bethesda). 2017;32:141-151.
2. Chubb SA, Davis WA, Inman Z, et al. Prevalence and progression of subclinical hypothyroidism in women with type 2 diabetes: the Fremantle Diabetes Study. Clin Endocrinol (Oxf). 2005;62:480-486.
3. Gyawali P, Takanche JS, Shrestha RK, et al. Pattern of thyroid dysfunction in patients with metabolic syndrome and its relationship with components of metabolic syndrome. Diabetes Metab J. 2015;39:66-73.
4. Lin SY, Wang YY, Liu PH, et al. Lower serum free thyroxine levels are associated with metabolic syndrome in a Chinese population. Metabolism. 2005;54:1524-1528.
5. Roos A, Bakker SJ, Links TP, et al. Thyroid function is associated with components of the metabolic syndrome in euthyroid subjects. J Clin Endocrinol Metab. 2007;92:491-496.
6. Park HT, Cho GJ, Ahn KH, et al. Thyroid stimulating hormone is associated with metabolic syndrome in euthyroid postmenopausal women. Maturitas. 2009;62:301-305.
7. Garduño-Garcia Jde J, Alvirde-Garcia U, López-Carrasco G, et al. TSH and free thyroxine concentrations are associated with differing metabolic markers in euthyroid subjects. Eur J Endocrinol. 2010;163:273-278.
8. Ruhla S, Weickert MO, Arafat AM, et al. A high normal TSH is associated with the metabolic syndrome. Clin Endocrinol (Oxf). 2010;72:696-701.
9. Lee YK, Kim JE, Oh HJ, et al. Serum TSH level in healthy Koreans and the association of TSH with serum lipid concentration and metabolic syndrome. Korean J Intern Med. 2011;26:432-439.
10. Oh JY, Sung YA, Lee HJ. Elevated thyroid stimulating hormone levels are associated with metabolic syndrome in euthyroid young women. Korean J Intern Med. 2013;28:180-186.
11. Mehran L, Amouzegar A, Tohidi M, et al. Serum free thyroxine concentration is associated with metabolic syndrome in euthyroid subjects. Thyroid. 2014;24:1566-1574.
12. Hage M, Zantout MS, Azar ST. Thyroid disorders and diabetes mellitus. J Thyroid Res. 2011;2011:439463.
13. Kalra S, Aggarwal S, Khandelwal D. Thyroid dysfunction and type 2 diabetes mellitus: screening strategies and implications for management. Diabetes Ther. 2019;10:2035-2044.
14. Vyakaranam S, Vanaparthy S, Nori S, et al. Study of insulin resistance in subclinical hypothyroidism. Int J Health Sci Res. 2014;4:147-153.
15. Irace C, Carallo C, Scavelli FBet al. Markers of insulin resistance and carotid atherosclerosis. A comparison of the homeostasis model assessment and triglyceride glucose index. Int J Clin Pract. 2013;67:665-672.
16. Unger G, Benozzi SF, Perruzza F, et al. Triglycerides and glucose index: a useful indicator of insulin resistance. Endocrinol Nutr. 2014;61:533-540.
17. Mohd Nor NS, Lee S, Bacha F, et al. Triglyceride glucose index as a surrogate measure of insulin sensitivity in obese adolescents with normoglycemia, prediabetes, and type 2 diabetes mellitus: comparison with the hyperinsulinemic-euglycemic clamp. Pediatr Diabetes. 2016;17:458-465.
18. Lee DY, Lee ES, Kim JH, et al. Predictive value of triglyceride glucose index for the risk of incident diabetes: a 4-year retrospective longitudinal study. PLoS One. 2016;11:e0163465.
19. Kitae A, Hashimoto Y, Hamaguchi M, et al. The triglyceride and glucose index is a predictor of incident nonalcoholic fatty liver disease: a population-based cohort study. Can J Gastroenterol Hepatol. 2019;2019:5121574.
20. Barzegar N, Tohidi M, Hasheminia M, et al. The impact of triglyceride-glucose index on incident cardiovascular events during 16 years of follow-up: Tehran Lipid and Glucose Study. Cardiovasc Diabetol. 2020;19:155.
21. Tagami T, Tamanaha T, Shimazu S, et al. Lipid profiles in the untreated patients with Hashimoto thyroiditis and the effects of thyroxine treatment on subclinical hypothyroidism with Hashimoto thyroiditis. Endocr J. 2010;57:253-8.
22. Mikos H, Mikos M, Rabska-Pietrzak B, et al. The clinical role of serum concentrations of selected cytokines: IL-1β, TNF-α and IL-6 in diagnosis of autoimmune thyroid disease (AITD) in children. Autoimmunity. 2014;47:466-472.
23. Contreras-Haro B, Hernandez-Gonzalez SO, Gonzalez-Lopez L, et al. Fasting triglycerides and glucose index: a useful screening test for assessing insulin resistance in patients diagnosed with rheumatoid arthritis and systemic lupus erythematosus. Diabetol Metab Syndr. 2019;11:95.
24. Choi W, Park JY, Hong AR, et al. Association between triglyceride-glucose index and thyroid function in euthyroid adults: The Korea National Health and Nutritional Examination Survey 2015. PLoS One. 2021;16:e0254630.
25. Lei Y, Yang J, Li H, et al. Changes in glucose-lipid metabolism, insulin resistance, and inflammatory factors in patients with autoimmune thyroid disease. J Clin Lab Anal. 2019;33:e22929.

26. Yuan Y, Chen S, Lin C, et al. Association of triglyceride-glucose index trajectory and frailty in urban older residents: evidence from the 10-year follow-up in a cohort study. Cardiovasc Diabetol. 2023;22:264.
27. Consitt LA, Clark BC. The vicious cycle of myostatin signaling in sarcopenic obesity: myostatin role in skeletal muscle growth, insulin signaling and implications for clinical trials. J Frailty Aging. 2018;7:21-27.
28. Akyüzlü ZN, Mutlu HH. Tiroit hastalığı olanlarda trigliserit/glikoz indeksi insülin direnci belirteci olarak kullanılabilir mi?. ETD. 2020;59:165-173.
29. Dessein PH, Joffe BI, Veller MG, et al. Traditional and nontraditional cardiovascular risk factors are associated with atherosclerosis in rheumatoid arthritis. J Rheumatol. 2005;32:435-442.
30. Sinicato NA, da Silva Cardoso PA, Appenzeller S. Risk factors in cardiovascular disease in systemic lupus erythematosus. Curr Cardiol Rev. 2013;9:15-19.
31. Pereira RM, de Carvalho JF, Bonfá E. Metabolic syndrome in rheumatological diseases. Autoimmun Rev. 2009;8:415-419.
32. Mazaheri T, Sharifi F, Kamali K. Insulin resistance in hypothyroid patients under Levothyroxine therapy: a comparison between those with and without thyroid autoimmunity. J Diabetes Metab Disord. 2014;13:103.
33. Biyikli HH, Arduc A, Isik S, et al. Assessing the relationship between serum ghrelin levels and metabolic parameters and autoimmunity in patients with euthyroid Hashimoto's thyroiditis. Endocr Pract. 2014;20:818-824.