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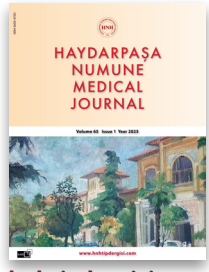

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ORIGINAL ARTICLE

The Relationship Between the Triglyceride-Glucose Index, and HbA1c and Insulin Resistance in Prediabetic Patients

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Abstract

Introduction: The triglyceride-glucose index (TyGi) is recommended as a reliable and simple insulin resistance (IR) marker in patients. This study aimed to clarify the role of TyGi in prediabetic patients and to compare its correlation with insulin resistance.

Methods: A total of 176 patients who applied to the Endocrinology Department outpatient clinic and underwent a 3-hour OGTT were included in the study. The patients were divided into two groups (normal, prediabetes) according to the OGTT results. IR was calculated using the homeostatic model assessment (HOMA-IR) formula. The ADA criteria were used to diagnose prediabetes. The formula ln (fasting triglyceride [mg/dL]×fasting glucose [mg/dL]/2) was used to determine the TyGi.

Results: Higher mean TyGi (8.79±0.08 vs. 8.43±0.05) and HOMA-IR (4.28±0.43 vs. 2.41±0.21) values were observed in prediabetic subjects compared to normoglycemic subjects. During the OGTT, TyGi correlated with glucose, HbA1c, and HOMA-IR measurements. The area under the curve (AUC) for HbA1c (0.754) was greater than that for HOMA-IR (0.725) and TyGi (0.674) in diagnosing prediabetes. The cut-off values for prediabetes were TyGi > 8.50 (sensitivity: 66.1%, specificity: 42.7%) and HOMA-IR > 2.25 (sensitivity: 62.5%, specificity: 32.6%).

Discussion and Conclusion: TyGi is nearly as effective as HbA1c as a diagnostic marker for prediabetes, and the cut-off points for identifying prediabetes were determined as TyGi > 8.50 and HOMA-IR > 2.25.

Keywords: HOMA-IR; insulin resistance; prediabetes; triglyceride glucose index.

Obesity, metabolic syndrome, and diabetes mellitus are chronic diseases with a rapidly increasing prevalence in our country and throughout the World[1-4]. Type 2 diabetes mellitus (DM) is a chronic disease that results from a combination of insulin resistance (IR) and impaired insulin secretion[5]. The gold standard for determining IR is the hyperinsulinemic-euglycaemic clamp, but it is impractical due to the difficulty of application. The homeostatic model assessment (HOMA) formula is a preferred method and is widely used in clinical settings to determine the risk of prediabetes and type 2 DM, fatty liver, acute coronary syndrome, and metabolic syndrome[6-8]. However, it cannot be used in patients who are receiving insulin therapy or those with low beta-cell reserve, and insulin is not routinely analyzed in many peripheral clinics.

The triglyceride-glucose index (TyGi) is a reliable and simple method for diagnosing IR in both adults and children. It is calculated using the formula ln (fasting triglyceride [mg/dL]×fasting glucose [mg/dL]/2)[9]. It has even been reported to be more sensitive than HOMA in diseases associated with insulin resistance[10-12]. Additionally, it has

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been claimed to be more sensitive than HOMA in assessing the likelihood that an adult or child may develop diabetes and in evaluating glycaemic control[13,14].

As part of this research, we aimed to clarify the role of the TyGi in prediabetic patients and to compare its correlation with insulin resistance.

Materials and Methods

Patients who applied to the outpatient endocrinology clinic and underwent a 3-hour OGTT were included in the study. The patients included in the study were separated into two groups (normal, prediabetes) according to the OGTT result. The protocols used for human experimentation were compliant with the 1975 Helsinki Declaration, revised in 2008, and the moral standards decided upon by national and institutional organizations. Patient data were retrospectively reviewed. Due to the retrospective design of our study, informed consent was not obtained. Ethics committee approval document date and number: 04/12/2023, 2023/211–4381.

IR was calculated using the homeostatic model assessment (HOMA-IR) formula[11]. ADA criteria were used to diagnose prediabetes. For the calculation of TyGi, the formula ln (fasting triglyceride [mg/dL]×fasting glucose [mg/dL]/2) was used[9].

Plasma glucose levels were measured by the hexokinase enzymatic method. In contrast, HDL cholesterol, LDL cholesterol, and triglycerides were measured by the colorimetric enzymatic test using the Roche/Hitachi cobas c system (Cobas C 702). Meanwhile, A1C was measured using Capillary Tera 3 via high-performance liquid chromatography. Serum insulin levels were measured by electrochemiluminescence immunoassay on a Cobas C immunochemistry analyser (Cobas C 601).

Statistical Analysis

Data collected in the study were analyzed using the IBM Statistical Package for the Social Sciences (SPSS) for Windows 18.0 (IBM Corp., Armonk, NY). As descriptive values, percentage and frequency were used for categorical data, and standard error of the mean (SEM) and mean were used for continuous data. Mann-Whitney U or t-tests were used to analyze the relationship between variables and groups. Pearson's correlation tests were used to examine the relationship between the TyGi index and clinical data. To evaluate the results, receiver operating characteristic (ROC) curves were generated. The performance of the predictors of prediabetes was determined using the area under the ROC curve (AUC–ROC). A p value of less than 0.05 was considered statistically significant.

Results

A total of 176 participants were admitted in the study. The gender distribution was 131 (74.4%) females and 45 (25.6%) males. The mean age of the patients was 43.90±1.04 years, ranging from 18 to 74 years. The mean HbA1c of the study cohort was 5.5±0.37%. A total of 59 participants (33.5%) showed prediabetes by OGTT. In addition, 49 participants (27.8%) had HbA1c levels between 5.7 and 6.4% (indicative of prediabetes).

The baseline biochemical and clinical demographic characteristics of the group are denoted in Table 1. Patients with prediabetes were older and more obese, with higher mean HbA1c and blood glucose levels, as expected. Lipid profiles were not significantly different. The comparison of the two groups is elaborated in Table 2.

Table 1. The clinical and biochemical characteristics of the group (n=176)

	Mean±SEM
Sex (n)	
Female	131 (74.4%)
Male	45 (25.6%)
Age (years)	43.90±1.05
BMI (kg/m^2)	32.99±1.08
Fasting glucose (mg/dL)	95.87±0.89
OGTT thirty-minute glucose (mg/dL)	153.08±25.4
OGTT one-hour glucose (mg/dL)	144.02±3.27
OGTT two-hour glucose (mg/dL)	108.08±2.34
OGTT three-hour glucose (mg/dL)	80.53±1.56
HOMA-IR	3.06±0.21
HbA1c (%)	5.51±0.03
Triglyceride glucose index	8.56±0.05
HDL cholesterol (mg/dL)	52.86±1.25
LDL cholesterol (mg/dL)	118.10±3.92
Triglyceride (mg/dL)	131.82±6.67
Mean OGTT (fasting and 2 h) (mg/dL)	101.97±1.44
Mean OGTT (fasting, 30 m,1 and 2 h) (mg/dL)	123.92±1.90
Mean OGTT (fasting, 30 m,1, 2 and 3 h) (mg/dL)	115.23±1.63
Impaired fasting glucose (n)	51 (30.2%)
Impaired glucose tolerance (n)	26 (15.4%)
Prediabetes HbA1c (n)	49 (31.8%)
Prediabetes OGTT (n)	59 (33.5%)

BMI: Body mass index; OGTT: Oral glucose tolerans test; HOMA-IR: Homeostatic model assessment for insuline resistance; HbA1c: Hemoglobin A1c; HDL cholesterol: High density lipoprotein cholesterol; LDL cholesterol: Low density lipoprotein cholesterol.

Table 2. Comparison of normal and prediabetic OGTT groups

	Normal OGTT (n=157) Mean±SEM	Prediabetes OGTT (n=59) Mean±SEM	p
Age (years)	40.77±1.31	50.10±1.44	<0.0001
BMI (kg/m^2)	31.08±1.24	37.08±1.75	=0.008
Fasting glucose (mg/dL)	89.39±0.62	107.95±1.20	<0.0001
OGTT thirty minute glucose (mg/dL)	144.27±2.84	171.98±4.23	<0.0001
OGTT one hour glucose (mg/dL)	128.45±3.12	177.53±5.65	<0.0001
OGTT two hour glucose (mg/dL)	96.08±2.10	130.44±4.12	<0.0001
OGTT three hour glucose (mg/dL)	76.88±1.71	87.83±2.99	=0.001
HOMA-IR	2.41±0.21	4.28±0.43	<0.0001
HbA1c (%)	5.41±0.03	5.71±0.05	<0.0001
Triglyceride glucose index	8.43±0.05	8.79±0.08	<0.0001
HDL cholesterol (mg/dL)	52.33±1.28	53.93±2.77	=0.548
LDL cholesterol (mg/dL)	115.11±4.72	124.23±7.02	=0.277
Triglyceride (mg/dL)	124.15±7.10	147.02±13.94	=0.106
Mean OGTT (fasting and 2 h) (mg/dL)	92.74±1.14	119.19±2.18	<0.0001
Mean OGTT (fasting, 30 m,1 and 2 h) (mg/dL)	114.31±1.71	144.54±3.07	<0.0001
Mean OGTT (fasting, 30 m,1, 2 and 3 h) (mg/dL)	92.74±1.14	119.19±2.18	<0.0001

BMI: Body mass index; OGTT: Oral glucose tolerans test; HOMA-IR: Homeostatic model assessment for insuline resistance; HbA1c: Hemoglobin A1c; HDL cholesterol: High density lipoprotein cholesterol; LDL cholesterol: Low density lipoprotein cholesterol.

Higher mean TyGi (8.79±0.08 vs. 8.43±0.05) and HOMA-IR (4.28±0.43 vs. 2.41±0.21) were observed in prediabetic subjects compared to normoglycaemic subjects. The TyGi index correlated with HOMA-IR, HbA1c, and glucose measurements during the OGTT (Table 3). TyGi index was higher in patients with prediabetes. The result was statistically significant. Similarly, HOMA-IR and HbA1c were also found to be significantly elevated. This analysis is presented in Table 2. The area under the curve (AUC) for HbA1c (0.754) was greater than that for HOMA-IR (0.725) and TyGi index (0.674) in diagnosing prediabetes. ROC curve is presented in Figure 1.

Table 3. Correlation of various parameters with triglyceride glucose index

Test result variables	r	P
Fasting glucose	0.289	<0.0001
OGTT thirty minute glucose	0.230	=0.005
OGTT one hour glucose	0.335	<0.0001
OGTT two hour glucose	0.306	<0.0001
OGTT three hour glucose	0.157	=0.47
HOMA-IR	0.293	<0.0001
HbA1c	0.299	<0.0001
Mean OGTT (fasting and 2 h)	0.425	<0.0001
Mean OGTT (fasting, 30 m,1 and 2 h)	0.391	<0.0001
Mean OGTT (fasting, 30 m,1, 2 and 3 h)	0.398	<0.0001

OGTT: Oral glucose tolerans test; HOMA-IR: Homeostatic model assessment for insuline resistance; HbA1c: Hemoglobin A1c.

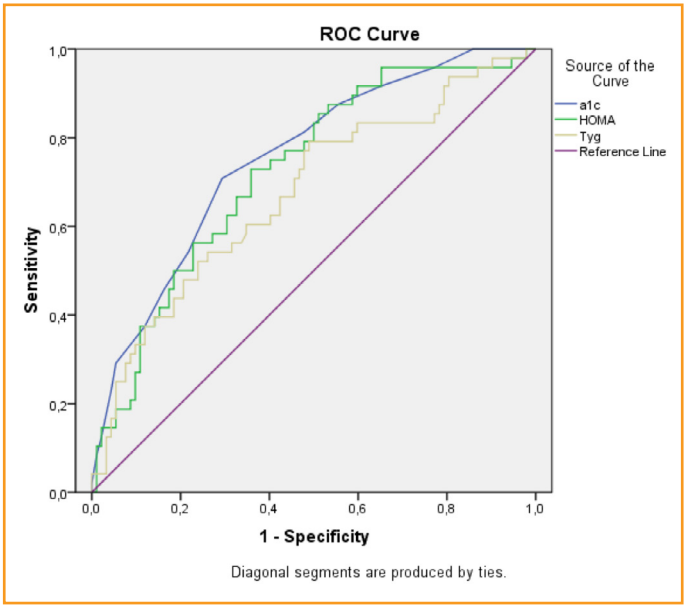

Figure 1. The area under the curve for HbA1C (0.754) was greater than that for HOMA-IR (0.725) and TyG index (0.674) in diagnosing prediabetes.

HOMA-IR: Homeostatic model assessment for insuline resistance; TyGi: The triglyceride-glucose index; AUC: Area under the curve; HbA1c: Hemoglobin A1c.

The cut-off values for prediabetes were TyGi>8.50 (with a sensitivity of 66.1% and specificity of 42.7%) and HOMA-IR>2.25 (with a sensitivity of 62.5% and specificity of 32.6%).

Discussion

Diabetes mellitus is a metabolic disease that increases the risk of early death due to its macrovascular and microvascular complications, as well as acute complications, and it has a high cost of care. Its incidence is increasing worldwide and varies across different populations. Atherosclerosis and clinical cardiovascular disease are directly or indirectly caused by high blood sugar. Cross-sectional studies have demonstrated that mild to moderate hyperglycemia, even below the diabetic threshold, increases the frequency of coronary disease[15]. A systematic review by Ford et al.[16] investigated the relationship between cardiovascular risk factors and prediabetes. A significant correlation between the TyGi index and the incidence of multivessel coronary artery disease was observed by Wang et al.[17]

Hyperglycemia is a well-defined cardiovascular risk factor. Although some evidence exists, the relationship between hyperglycemia, prediabetes, and vascular damage is complex. Even within the prediabetic range, hyperglycemia may increase inflammation, extracellular matrix thickening, impairment of endothelial nitric oxide activity, endothelial damage, atherosclerosis, pericyte loss, capillary microaneurysms, vascular proliferation, and vascular complications by stimulating the expression of vascular adhesion molecules and cytokines[18].

There are various methods used to measure IR. The hyperinsulinemic-euglycemic clamp method is the gold standard in IR measurement[19]. However, this method is costly and challenging to implement. Some mathematical formulas that measure insulin sensitivity and are easier to apply have been developed. The most commonly used is HOMA-IR, calculated based on fasting plasma glucose and insulin. Studies have found that IR measured by the hyperinsulinemic-euglycemic clamp technique is highly correlated with HOMA-IR[20-22]. However, insulin cannot be measured in every centre, particularly in primary care settings. For this reason, a basic, reliable, and cost-effective method of measuring IR was needed, and TyGi was developed by Simmental-Mendia et al.[23,24] The TyGi is calculated using fasting plasma glucose and triglycerides (TG)[23].

Some indices have been formulated to measure insulin resistance, using different lipid parameters in proportion. These include TG/HDL-C, LDL-C/HDL-C, apoB/apoA-I ratio, lipid accumulation product (LAP), TyGi, visceral adiposity index (VAI), etc. According to research, the TyGi, one of these indices, showed a strong correlation with HOMA-IR[23]. One study reported that "the TyGi correlated more with the hyperinsulinemic-euglycemic clamp test than HOMA-IR"[24]. A positive relationship between the TyGi and HbA1c was found in one research[25]. The relevance of the TyGi in predicting IR was attempted to be understood in a study where patients were identified using the hyperinsulinemic-euglycemic clamp test. This study's sensitivity (96.5%) and specificity (85%) in identifying insulin resistance were achieved when the cut-off point was set at 4.68[24]. In another study, when the cut-off point was taken as 4.65, the sensitivity of the TyGi in determining IR was found to be 84%, and the specificity was 45%[24]. Akyuzlu and Mutlu reported that the cut-off point value of the TyGi, which makes patients with IR predictable, was found to be 8.64. When the cut-off point value was taken as 8.64, the sensitivity of the TyGi index was found to be 60%[26].

Our research revealed that a higher mean TyGi (8.79±0.08 vs. 8.43±0.05) and HOMA-IR (4.28±0.43 vs. 2.41±0.21) were observed in prediabetic subjects compared to normoglycaemic subjects. HOMA-IR, HbA1c, and glucose measurements during the OGTT were correlated with the TyGi. The area under the curve (AUC) for HbA1c (0.754) was greater than that for HOMA-IR (0.725) and TyGi (0.674) in diagnosing prediabetes. The cut-off values for prediabetes were TyGi>8.50 (with a sensitivity of 66.1% and specificity of 42.7%) and HOMA-IR>2.25, indicating 62.5% sensitivity and 32.6% specificity.

A considerably higher HOMA index was found in participants with hyperlipidemia, central obesity, and diabetes in the author's recent study; in various subject groups, 2.24 indicated clinical and metabolic signs of IR[7].

The main goal of prediabetes is to avoid the development of diabetes. To achieve this outcome, beta cell function must be preserved, and microvascular and cardiovascular complications must be prevented or delayed. In addition to maintaining beta cell function, practices that alter insulin sensitivity determine the risk of developing type 2 diabetes. The most critical clinical data determining the risk of developing type 2 diabetes in prediabetic patients vary depending on the parameters used in the diagnosis of prediabetes. Individuals who have a family history of diabetes or obesity, in addition to other clinical risk factors for the disease, are more likely to develop diabetes. Yoon JS et al. found "TyGi index to be a superior biomarker for prediction of type 2 diabetes mellitus in children and adolescents"[14].

Conclusion

The findings of this study suggest that the TyGi index is nearly as effective as HbA1c as a diagnostic marker for prediabetes, and the cut-off points for identifying prediabetes were determined as TyGi>8.50 and HOMA-IR>2.25. The primary limitation of our study was the small sample size. Future studies with larger sample sizes may yield more robust analyses of the TyGi index.

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Data Availability: This article does not include any personal or patient data.

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