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The role of triglyceride-glucose index in determining subclinical atherosclerosis in patients with primary hypertension

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Abstract. – OBJECTIVE: With the current study, we aimed at examining the relationship between the triglyceride-glucose (TyG) index and subclinical atherosclerosis in patients with primary hypertension.

PATIENTS AND METHODS: 185 patients with primary hypertension were included in this study. The following findings were considered to be associated with target organ damage (TOD): urinary protein excretion > 150 mg/dL and microalbumin excretion > 30 mg/dL, carotid intima-media thickness (CIMT) ≥ 0.9 mm or carotid plaque and/or left ventricular mass index (LVMI) > 95 g/m² in women, > 115 g/m² in men.

RESULTS: TyG index values were positively correlated with levels of CIMT (r=0.434; $p<0.001$), LVMI (r=0.351; $p<0.001$), microalbuminuria (r=0.347; $p<0.001$), and proteinuria (r=0.355; $p<0.001$). In the multivariable regression model, in which the variables associated with the presence of TOD were included, increased age (OR: 1.04, $p=0.025$), increased body mass index (OR: 1.10, $p=0.042$), and increased TyG index value (OR: 1.05, $p<0.001$) had independent associations with TOD. The threshold value of the TyG index for the presence of TOD was determined as > 8.85 with 79.0% sensitivity and 77.1% specificity (AUC±SE: 0.859±0.03, +PV: 70.6%, -PV: 84.0%, $p<0.001$). The TyG index had a superior diagnostic discrimination compared to its components in predicting the presence of TOD.

CONCLUSIONS: Increased TyG index values in patients with primary hypertension are associated with damage to target organs, not merely subclinical atherosclerosis.

Key Words:
CIMT, End organ damage, Endothelial dysfunction, LVMI, TyG index.

Introduction

Primary hypertension is a clinical condition that plays a major role in the etiopathogenesis of atherosclerotic diseases[1]. As a result of increased blood pressure, damage occurs in the endothelium by both direct effects and shear stress[2,3]. Due to these factors, chronic inflammation and a serious increase in the level of reactive oxygen radicals occur in relation to the damage that begins in the endothelium[4-6]. All of these pathophysiological processes triggered by high blood pressure lead to an increase in endothelial dysfunction.

Endothelial dysfunction constitutes the basic initial step of atherosclerotic processes occurring in the cardiovascular and cerebrovascular systems[7]. Therefore, in order to prevent pathophysiological processes of atherosclerosis, it is necessary to determine damage beginning in the endothelium and take precautions accordingly. Imaging methods are generally used to determine endothelial dysfunction. The main ones are echocardiography to evaluate the left ventricular mass index (LVMI) and Doppler ultrasonography to evaluate the carotid intima-media thickness (CIMT)[8]. However, these examinations cannot be performed frequently because they are not available in every center and a health professional is needed for their application. Therefore, easily measurable parameters are needed to determine endothelial dysfunction in the subclinical stage of atherosclerosis.

In recent studies[9], it has been determined that the triglyceride-glucose (TyG) index, which can be calculated using fasting blood glucose and triglyceride levels, is a reliable indicator of insulin resistance. Insulin resistance is known to be one of the clinical conditions that play roles in the pathophysiology of atherosclerosis in both diabetes and prediabetic processes[10,11]. Therefore, it has been shown in several studies[12] that the TyG index is a parameter associated with cardiovascular diseases. At the same time, it has been shown in other studies[13,14] that the TyG index is associated with high blood pressure. However, there are very few studies[15,16] examining the relationship of the TyG index with subclinical atherosclerosis in cases of primary hypertension.

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Therefore, in this study we aimed at examining the role of the TyG index in determining subclinical atherosclerosis in primary hypertension patients.

Patients and Methods

This study was performed retrospectively in Ankara City Hospital's Internal Medicine Clinic between May 15 and June 15, 2022. The study was designed in accordance with the Declaration of Helsinki. Ethics committee approval was obtained for the study by the Ankara City Hospital.

Patients aged > 18 years who had been examined in detail upon admission to our clinic and followed with the diagnosis of primary hypertension were evaluated for this study. Among these patients, those who had CIMT and/or LVMI measurements recorded as indicators of target organ damage (TOD) were enrolled in the study.

Patients with known cardiovascular disease, cerebrovascular disease, secondary hypertension, nephrotic syndrome, rheumatic disease, malignancy, acute liver or kidney failure, chronic liver or kidney failure, or active infection and patients who were not evaluated for TOD were excluded from the study.

Patients were divided into two groups, as those with and without target organ damage. Those with CIMT ≥ 0.9 mm or carotid plaque and/or LVMI of > 95 g/m^2 in women and > 115 g/m^2 in men were considered as patients with TOD[17]. Urinary protein excretion of > 150 mg/dL and microalbumin excretion of > 30 mg/dL were also considered to be associated with TOD[8,18].

The clinical demographic findings, laboratory findings, and imaging methods of the study population were obtained from the patients' electronic files. Biochemical parameters were obtained from venous blood samples taken during evaluations in the outpatient clinic after 12 hours of fasting. Lipid parameters and glucose levels of all patients were evaluated in the same laboratory with the same methods. The TyG index was calculated with the following formula: Ln [triglyceride (mg/dL) × glucose (mg/dL)/2].

Biochemical Analysis

For the analysis of biochemical parameters, blood samples were taken from the participants after 12 hours of fasting. Biochemical analysis was performed with kits of the same brand in the same laboratory.

A 24-hours urine sample was requested from all patients. How they had be collected was explained to the patients by the laboratory staff. While 24-hours urinary protein was determined by the microalbumin turbidimetric method, total protein was determined by albumin colorimetric method and uric acid, fasting glucose, triglyceride, and total cholesterol were determined by enzymatic colorimetric method. High-density lipoprotein (HDL) cholesterol was determined by the homogeneous enzymatic colorimetric method using a Hitachi Modular P800 autoanalyzer (Roche Diagnostics Corp., Indianapolis, IN, USA). Low-density lipoprotein (LDL) cholesterol was calculated using the Friedewald method.

Echocardiographic Examination

Echocardiography was performed by cardiologists who were unaware of the clinical status of the patients, using the GE Vivid 7 device (GE Vingmed Ultrasound AS, Horten, Norway). Two-dimensional, color, pulsed, and continuous wave Doppler examinations were performed using standard techniques. The Devereux formula was used to calculate the LVMI[19] as follows: LVMI = 1.04 × [(IVST + PWT + LVDd)3 – (LVDd)3] – 13.6.

Carotid Ultrasonography Evaluation

CIMT was measured by radiologists who were unaware of the clinical status of the patients, using a high-resolution B-mode ultrasonography device (Logiq 7, GE Healthcare, Chicago, IL, USA). The mean CIMT was calculated by taking the averages of 3 measurements for each carotid artery. The presence of plaque in the carotid artery was evaluated and recorded[20].

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics 26 for Windows (IBM Corp., Armonk, NY, USA). The extent to which the data followed a normal distribution was evaluated using the Kolmogorov-Smirnov test. Numeric parameters with normal distribution were presented as mean ± standard deviation (SD) and numeric parameters without normal distribution were presented as median (interquartile range: IQR). Correlations between numeric parameters were analyzed *via* Pearson and Spearman correlation analysis. Numeric variables with and without normal distribution were plotted as mean ± standard deviation and median (25th and 75th IQRs), respectively. Cate-

gorical variables were indicated as numeric and percentile values. Chi-square, Yates correction, and Fisher's exact tests were used for comparisons of categorical data. The Student's *t*-test or Mann-Whitney U test were used for the comparison of numeric variables between the two groups according to the distribution of normality. ANOVA (post-hoc: Bonferroni test) or the Kruskal-Wallis' H test (post-hoc: Dunn test) were used for comparisons of numeric variables between the TyG index quartile groups according to the normality distribution. Stepwise multivariable logistic regression analysis was used to predict TOD. Diagnostic performance evaluation of the TyG index was performed with receiver operating characteristic (ROC) curve analysis and the threshold values were determined according to the Youden index method. Values of $p<0.05$ were considered to be significant in statistical analyses.

Results

This study included 185 patients, 76 of whom had TOD, while 109 patients did not. The mean age of the patients was 52.5±12.0 years and the majority of them were women (70.8%). In patients with primary hypertension, the TyG index positively correlated with the values of CIMT (r=0.434; $p<0.001$) (Figure 1A), LVMI (r=0.351; $p<0.001$) (Figure 1B), microalbuminuria (r=0.347; $p<0.001$) (Figure 1C), and proteinuria (r=0.355; $p<0.001$) (Figure 1D) (Table I).

The mean age (57.4±9.2 *vs.* 49.2±12.5 years; $p<0.001$), proportion of female patients (78.9% *vs.* 65.1%; $p=0.042$), median duration of hypertension (5 *vs.* 3; $p=0.008$), mean glucose level (101.0±13.0 *vs.* 95.6±13.9; $p=0.009$), mean total cholesterol level (211.8±44.4 *vs.* 197.5±37.8; $p=0.020$), median triglyceride level (182 vs. 120; $p<0.001$), mean TyG index value (9.1±0.4 vs. 8.6±0.4; $p<0.001$) (Figure 2A), and median CRP level (4.3 *vs.* 3; $p=0.047$) were higher among patients with TOD compared to those without, while the mean HDL level was lower (46.0±13.5 *vs.* 52.5±14.2; $p=0.002$) (Table II). In the multivariable regression model, in which the variables associated with the presence of TOD were included, increased age (OR: 1.04, $p=0.025$), increased BMI (OR: 1.10, $p=0.042$), and increased TyG index (OR: 1.05, $p<0.001$) had independent associations with TOD. According to these findings, it was determined that a 1-unit increase in the TyG index increased the probability of TOD by 1.05-fold (Table III). The threshold value of the TyG index for the presence of TOD was determined as > 8.85 with 79.0% sensitivity and 77.1% specificity (AUC±SE: 0.859±0.03, +PV: 70.6%, -PV: 84.0%, $p<0.001$). The TyG index had superior diagnostic discrimination compared to its components in predicting the presence of TOD (Figure 2B).

We also analyzed the distribution of demographic and laboratory findings according to the quartiles of the TyG index. As shown in Table IV, the TyG index was positively associated with LVMI and microalbuminuria. Median proteinuria and mean CIMT levels were similar in the Q3 and Q4 groups, and those values were higher than the values of the other quartiles. Mean age and median CRP levels were higher in the Q4 group compared to the other quartiles (Table IV).

Discussion

In our study, we examined whether the TyG index was a marker associated with subclinical atherosclerosis in patients with primary hypertension. There was a positive correlation between the TyG index and CIMT, LVMI, urinary protein, and urinary microalbumin levels. Multivariable regression analysis showed that an increase in the TyG index had an independent association with the presence of TOD.

Endothelial dysfunction due to primary hypertension constitutes one of the first steps of the atherosclerotic process. For this reason, events that start in the endothelium should be detected at subclinical levels in order to avoid any clinically evident events in the vascular system. LVMI, CIMT, retinopathy, and urinary protein excretion levels are frequently used as indicators of endothelial dysfunction. However, most of these examinations require specialized equipment and a professional team. For this reason, rapid, easily measurable, and inexpensive markers are needed to reveal subclinical damage to the endothelium due to primary hypertension.

Insulin resistance is among the important clinical conditions that play roles in the pathophysiology of atherosclerosis, and the TyG index is a reliable indicator of insulin resistance[9-11]. Insulin resistance is associated with macro- and microvascular damage[21]. It has been suggested[22] that fasting glucose reflects insulin resistance from the liver, while fasting

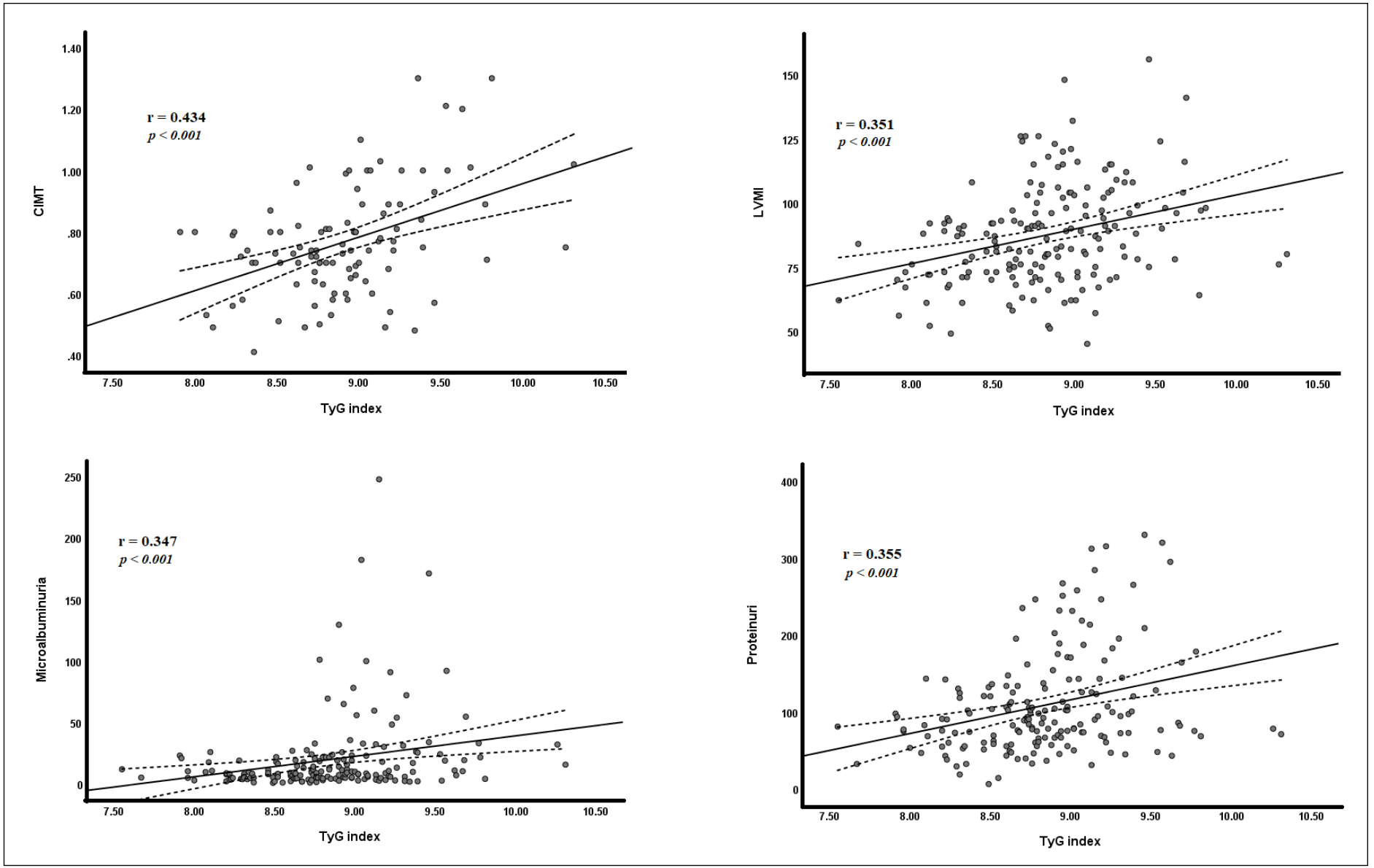

Figure 1. Relationship between TyG index and indicators of TOD.

Table I. Factors associated with target organ damage in patients with hypertension.

Variables	CIMT		LVMI		Microalbuminuria		Proteinuria	
	r	p	r	p	r	p	r	p
LVMI	0.251	0.045*	-	-	-	-	-	-
Microalbuminuria	0.270	0.033*	0.302	0.013*	-	-	-	-
Proteinuria	0.030	0.771	0.105	0.179	0.549	<0.001*	-	-
Age	0.532	<0.001*	0.322	<0.001*	0.078	0.309	-0.027	0.725
BMI	0.232	0.022*	0.025	0.748	0.110	0.154	0.147	0.054
Glucose	0.190	0.060	0.142	0.063	-0.010	0.899	0.014	0.8
Total cholesterol	0.109	0.284	0.057	0.455	0.052	0.492	0.019	0.805
LDL	-0.036	0.725	0.057	0.454	-0.011	0.890	-0.039	0.612
HDL	-0.111	0.273	-0.283	0.016*	-0.279	0.018*	-0.277	0.037*
Triglyceride	0.361	<0.001*	0.301	<0.001*	0.327	<0.001*	0.308	<0.001*
TyG index	0.434	<0.001*	0.351	<0.001*	0.347	<0.001*	0.355	<0.001*
Total protein	-0.046	0.657	0.050	0.519	0.094	0.219	-0.017	0.828
Albumin	-0.160	0.115	-0.031	0.684	0.085	0.265	-0.037	0.627
CRP	0.156	0.136	0.068	0.384	0.063	0.420	0.063	0.418

*$p < 0.05$ indicates statistical significance. BMI, body mass index; CIMT, carotid intima-media thickness; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVMI, left ventricular mass index; TyG, Triglyceride-glucose.

triglyceride reflects insulin resistance from adipose cells. The TyG index can reflect insulin resistance in both directions. Therefore, the TyG index can be an important indicator of events closely related to insulin resistance, such as oxidative stress, endothelial dysfunction, and kidney damage[23,24]. Many studies[12-14] have shown that the TyG index is associated with insulin resistance-related hypertension and cardiovascular diseases. In a study by Zheng and Mao[13], 4,686 healthy participants were followed for 9 years. Hypertension developed in 43.7% of these individuals. In Cox regression analysis, it was determined that the increased incidence of hypertension was associated with high TyG index values. In a community-based study by Bala et al[25], a significant relationship was found between the TyG index and the presence of hypertension. Likewise, in a community-based study conducted by Wang et al[26] in China, it was determined that increased TyG index values were associated with a high risk of prehy-

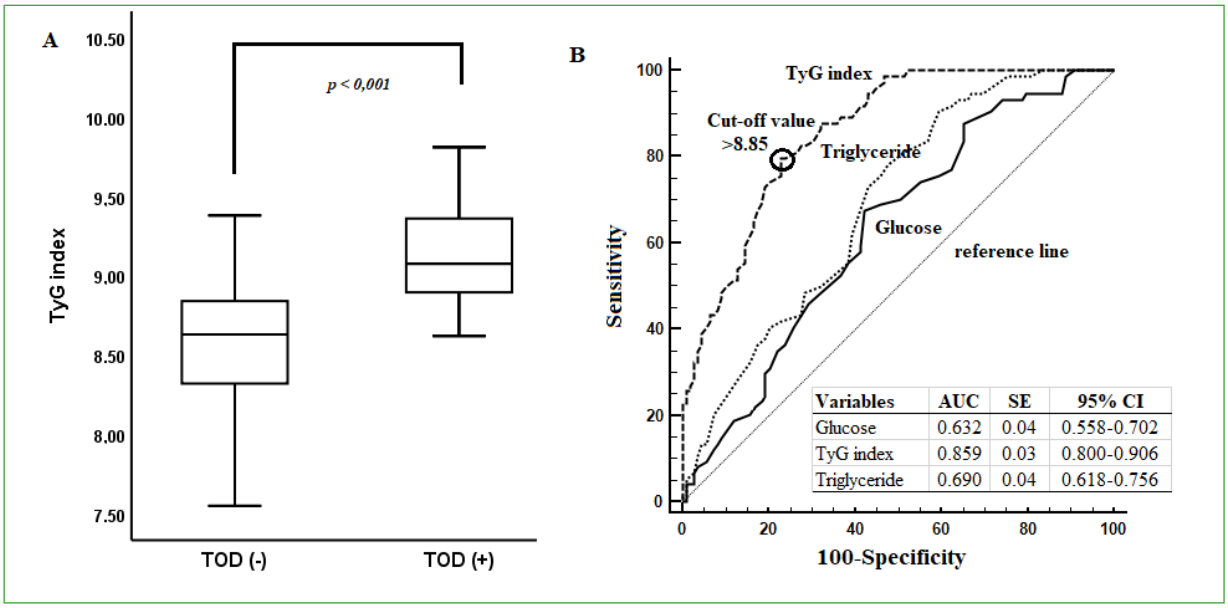

Figure 2. Distribution of TyG index (**A**) and threshold value (**B**) according to the presence of target organ damage.

Table II. Distribution of demographic and laboratory findings regarding the presence of target organ damage.

Variables	All population n=185	Target organ damage		P
		No n=109	Yes n=76	
Age, years	52.5±12.0	49.2±12.5	57.4±9.2	<0.001*
Gender, n (%)				
Female	131 (70.8)	71 (65.1)	60 (78.9)	0.042*
Male	54 (29.2)	38 (34.9)	16 (21.1)	
BMI, kg/m^2	29.8±4.7	28.8±4.2	31.1±5	0.001*
Current Smoker, n (%)	21 (11.4)	15 (13.8)	6 (7.9)	0.247
Duration of HT	3 (2-6)	3 (2-5)	5 (2-7)	0.008*
Drugs, n (%)				
ACE/ARB	101 (54.6)	57 (52.3)	44 (57.9)	0.452
Beta blocker	23 (12.4)	10 (9.2)	13 (17.1)	0.118
CCB	71 (38.4)	42 (38.5)	29 (38.2)	0.999
Diuretics	66 (35.7)	36 (33.0)	30 (39.5)	0.368
ASA	4 (2.2)	2 (1.8)	2 (2.6)	0.990
CIMT, mm	0.8±0.2	0.7±0.1	0.9±0.2	<0.001*
LVMI, g/m^2	87.5±19.7	79.2±13.6	98.8±21.3	<0.001*
Microalbuminuria, mg/24 h	9.2 (4.9-21.0)	6.8 (4.1-11.5)	19.1 (9.5-34.7)	<0.001*
Proteinuria, mg/24 h	90 (66.3-131.9)	76.0 (55.2-101.1)	135.3 (78.0-203.8)	<0.001*
Retinopathy, n (%)				
None	103 (55.7)	64 (58.7)	39 (51.3)	<0.001*
Grade I	46 (24.9)	29 (26.6)	17 (22.4)	
Grade II	32 (17.3)	16 (14.7)	16 (21.1)	
Grade III	4 (2.2)	-	4 (5.3)	
Glucose, mg/dL	97.8±13.7	95.6±13.9	101.0±13.0	0.009*
Total cholesterol, mg/dL	203.3±41.1	197.5±37.8	211.8±44.4	0.020*
LDL, mg/dL	122.9±36.1	121.8±34.8	124.4±37.9	0.629
HDL, mg/dL	49.8±14.2	52.5±14.2	46.0±13.5	0.002*
Triglyceride (mg/dl)	141 (108.5-188.0)	120 (89.5-147.5)	182 (142.3-232.0)	<0.001*
TyG index	8.8±0.5	8.6±0.4	9.1±0.4	<0.001*
Total protein, g/L	75.9±3.7	75.9±3.9	75.8±3.4	0.819
Albumin, g/L	46.3±2.8	46.5±2.9	46.2±2.7	0.479
CRP, mg/dL	3.2 (1.5-5.7)	3 (1.2-4.9)	4.3 (2.1-6.7)	0.047*

Data are mean±standard deviation or median (IQR), or number (%). *: $p < 0.05$ indicates statistical significance. ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ASA, acetylsalicylic acid; BMI, body mass index; CCB, calcium channel blockers; CIMT, carotid intima-media thickness; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVMI, left ventricular mass index; TyG, Triglyceride-glucose.

pertension and hypertension. These studies support the idea that high TyG index values may be associated with high blood pressure.

Many studies[27,28] have revealed that the TyG index may be associated with cardiovascular diseases. In a study by Park et al[28], healthy individuals without any comorbidities or smoking history were evaluated. In that study, the TyG index was determined to be an independent marker predicting subclinical coronary artery disease in traditionally healthy individuals. Baydar et al[27] examined a healthy population of 1,095 people and found a positive correlation between pulse wave velocity and TyG index values in those individuals. In different studies, the TyG index was found to be associated with the development of myocardial infarction[29], the development of stroke[30], the development of peripheral artery disease[31], and the risk of ischemic heart disease[32].

There is a limited number of studies[33] in literature examining the relationship between the TyG index and subclinical atherosclerosis in hypertensive patients. In a study by Cetin Sanlialp et al[33], in newly diagnosed hypertension patients, high TyG index values were found to be associated with left ventricular diastolic dysfunction and structural abnormalities. In our study, TyG index values were found to be higher in hypertension patients with TOD compared to those without. In correlation analysis, a positive correlation was found between the TyG index and LVMI, CIMT, proteinuria, and microalbuminuria. In multivariate regression analysis, the TyG index was found to be a risk factor asso-

Table III. Parameters independently associated with target organ damage.

Variables	Univariable Regression				Multivariable Regression			
	OR	95% CI		p	OR	95% CI		p
		lower	upper			lower	upper	
Age	1.07	1.04	1.10	<0.001*	1.04	1.01	1.07	0.025*
Gender								
Female	2.01	1.02	3.95	0.044*				
Male	ref							
BMI	1.12	1.04	1.20	0.002*	1.10	1.01	1.21	0.042*
Duration of HT	1.08	1.01	1.16	0.026*				
Glucose	1.03	1.01	1.05	0.011*				
Total cholesterol	1.01	1.00	1.02	0.022*				
HDL	0.96	0.94	0.99	0.003*				
Triglyceride	1.03	1.02	1.04	<0.001*				
TyG index	1.05	1.03	1.06	<0.001*	1.05	1.03	1.06	<0.001*
CRP	1.06	1.01	1.13	0.048*				
					Nagelkerke R^2= 0.564, p<0.001			

*p < 0.05 indicates statistical significance. BMI, body mass index; CRP, C-reactive protein; HDL, high-density lipoprotein; TyG, Triglyceride-glucose.

ciated with TOD. Considering these results, the TyG index, which can be easily and cheaply calculated in almost every center, may be a good marker that can show subclinical atherosclerosis in patients with hypertension.

When the data in the literature were examined, it was seen that the TyG index is associated with many clinical conditions. This information made us think that the relationship between the TyG index and subclinical atherosclerosis in hypertensive patients may have low sensitivity and specificity. Therefore, we applied ROC curve analysis and found that if the TyG index value is greater than 8.85, TOD is predicted with 79% sensitivity and 77.1% specificity. These findings suggest that the TyG index is an acceptable index for predicting TOD in cases of hypertension.

Limitations

The retrospective nature of our study is one of its main limitations. However, the negative impact of this limitation on the study is minimal, since both target organ parameters and other clinical and demographic findings of the participants included in the study were recorded in detail. Another limitation is that several parameters were used for the target organ evaluations of the patients. According to the parameters used, it was revealed whether there was TOD or not.

Conclusions

There was a positive relationship between the TyG index and TOD indicators in hypertensive patients, and multivariate regression analysis showed that increasing TyG index values had an independent relationship with the presence of TOD. Therefore, increased TyG index values in patients with primary hypertension are associated with damage to target organs, not merely subclinical atherosclerosis. These findings indicate that the TyG index may be an inexpensive, noninvasive, and easily measurable screening tool for TOD in hypertensive patients. However, prospective studies are needed to confirm the use of the TyG index as a routine subclinical atherosclerosis parameter in clinical practice.

Conflicts of Interest

The authors declared no conflict of interest.

Ethics Approval

Ankara City Hospital Ethics Committee, Decision Date/No.: 06.2022/E2-22-2036. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013.

Table IV. Distribution of demographic and laboratory findings by TyG index values.

Variables	Q1 (<8.58) n=46	Q2 (8.59-8.82) n=47	Q3 (8.83-9.13) n=45	Q4 (>9.14) n=47	p
Age, years	**46.7±13.2**	52.8±12.8	53.0±10.6	**57.6±9.0**	0.001*
Gender, n (%)					
Female	33 (71.7)	33 (70.2)	31 (68.9)	34 (72.3)	0.988
Male	13 (28.3)	14 (29.8)	14 (31.1)	13 (27.7)	
BMI, kg/m^2	29.1±4.5	29.1±4.9	29.5±3.8	31.3±5.0	0.073
Current Smoker, n (%)	1 (2.2)	9 (19.1)	9 (20.0)	2 (4.3)	0.004*
Duration of HT, years	2 (2-5)	3 (1-6)	4 (2-6)	5 (2-9)	0.091
Drugs, n (%)					
ACE/ARB	24 (52.2)	21 (44.7)	26 (57.8)	30 (63.8)	0.286
Beta blocker	7 (15.2)	2 (4.3)	8 (17.8)	6 (12.8)	0.171
CCB	13 (28.3)	21 (44.7)	20 (44.4)	17 (36.2)	0.306
Diuretics	16 (34.8)	17 (36.2)	14 (31.1)	19 (40.4)	0.834
ASA	0	1 (2.1)	2 (4.4)	1 (2.1)	0.474
CIMT, mm	0.7±0.1	0.7±0.1	**0.9±0.2**	**0.9±0.2**	0.001*
LVMI, g/m^2	**77.3±12.8**	**85.3±16.8**	**90.2±15.3**	**96.5±16.2**	0.001*
Microalbuminuria, mg/24 h	**7.7 (4.1-10.6)**	**8.4 (4.5-17.7)**	**11.7 (5.8-23.7)**	**17 (5.8-32.2)**	0.001*
Proteinuria, mg/24 h	73.8 (52.4-101.7)	86.6 (60.3-107.9)	**102.2 (73.3-171)**	**100.4 (72.5-182.5)**	<0.001*
Retinopathy, n (%)					
None	27 (58.7)	30 (63.8)	22 (48.9)	24 (51.1)	
Grade I	11 (23.9)	11 (23.4)	11 (24.4)	13 (27.7)	
Grade II	8 (17.4)	6 (12.8)	11 (24.4)	7 (14.9)	0.563
Grade III	0	0	1 (2.2)	3 (6.4)	
Glucose, mg/dL	92.2±13.1	96.0±12.1	97.6±11.4	**105.3±15**	<0.001*
Total cholesterol, mg/dL	180.4±31.6	211.0±36.8	197.8±35.1	**223.4±47.3**	<0.001*
LDL, mg/dL	107.6±30.1	131.3±32.3	122.5±35.6	129.8±41.3	0.005*
HDL, mg/dL	57.5±17.2	53.4±11.3	**45.1±10.3**	**43.2±12.5**	<0.001*
Triglyceride (mg/dl)	**85.5 (74-97)**	**126 (114-138)**	**155 (144-175)**	**219 (200-272)**	<0.001*
TyG index	**8.3±0.2**	**8.7±0.1**	**9.0±0.1**	**9.4±0.3**	<0.001*
Total protein, g/L	75.6±4.1	76.1±3.9	75.3±2.8	76.4±3.8	0.508
Albumin, g/L	46.4±2.7	46.6±2.4	46.4±2.7	45.9±3.3	0.679
CRP, mg/dL	2.8 (1.2-5)	2.8 (1.4-5)	2.8 (1.6-5.4)	**3.8 (2.4-6.7)**	0.044*

*$p < 0.05$ indicates statistical significance. Bolded characters show differences between groups. ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; ASA, acetylsalicylic acid; BMI, body mass index; CCB, calcium channel blockers; CIMT, carotid intima-media thickness; CRP, C-reactive protein; HDL, high-density lipoprotein; LDL, low-density lipoprotein; LVMI, left ventricular mass index; TyG, Triglyceride-glucose.

Informed Consent

Informed consent was obtained from all participants included in the study.

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Authors' Contribution

Concept –O.I., E.S.S. and İ.A.; Design – O.I., E.S.S. and İ.A.; Supervision – O.İ., E.S.S. and I.A.; Data collection &/or processing – E.S.S. and O.I.; Analysis &/or interpretation – O.I., E.S.S. and I.A.; Literature search – O.I., E.S.S. and I.A.;Data collection &/or processing – O.I., E.S.S. and I.A.; Writing – E.S.S.; Critical review – O.I., E.S.S. and I.A.

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