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Association Between Triglyceride-Glucose Index and Heart Rate Recovery Affecting Circadian Rhythm of Blood Pressure in Patients with Normoglycemic Primary Hypertension

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Abstract

Objectives: Although the cause of the disturbance of circadian blood pressure (BP) variation is not fully understood, insulin resistance (IR) and autonomic dysfunctions are thought to play a role. This study aimed to evaluate the relationships between the triglyceride-glucose (TyG) index, as a reliable surrogate for IR, and cardiac autonomic function as measured using heart rate (HR) recovery (HRR) levels according to circadian rhythm types of BP in patients with normoglycemic primary hypertension (NPHT).

Materials and Methods: This retrospective study included 254 patients with NPHT patients. The definition of non-dipper BP pattern included a <10% decline in night-time BP. The HRR levels were analyzed by subtracting HRs at 1-3 min from the maximal HR recorded during stress tests.

Results: Mean TyG index was higher in the non-dipper group than the dipper group (8.9±0.5 vs. 8.5±0.6, p<0.001), while mean HRR1 (26.8±8.7 vs. 31.7±9.6 bpm, p<0.001) value was lower. There was a negative correlation between HRR1 and TyG index (r=-0.316, p<0.001) and the blunted decline in night-time BP (r=-0.328, p<0.001). TyG index (β ±

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SE=-0.55±0.20, p<0.001) and HRR1 (β ± SE=0.58±0.14, p<0.001) levels were determined as independent predictors of blunted decline in night-time BP.

Conclusion: The elevated TyG index was associated with belated recovery of HR and it was an important predictor of blunted declines in night-time BP. Patients with NPHT may be at a risk of autonomic dysfunction due to increased IR.

Keywords: Blood pressure, circadian rhyth, heart rate, insulin, hypertension

Introduction

Blood pressure (BP) levels vary throughout the day, depending on the circadian rhythm of metabolism[1]. A decrease of less than 10% in BP levels compared to daytime values during sleep is defined as a non-dipper pattern (NDP)[2]. It is known that NDP increases the risk of organ damage and cardiovascular (CV) events in patients with hypertension[3,4].

Although the cause of the disturbance in the circadian BP variation is not fully understood, insulin resistance (IR) and autonomic dysfunctions are thought to play a role[5,6]. Patients suffering from NDP are prone to impaired autonomic functions, including sympathetic or parasympathetic activities[7]. Heart rate (HR) recovery (HRR), an easy and non-invasive indicator of autonomic function, is defined as the HR reduction after exercise. Delayed HRR is an important indicator of major CV events[8,9]. Increasing evidence suggested a significant association between HRR and circadian BP patterns[10,11].

The triglyceride-glucose (TyG) index, a surrogate marker of IR, has been implicated as a newly identified CV risk factor. IR may be involved in the pathogenesis of hypertension and autonomic nervous system dysfunction by mediating low-grade systemic inflammation[12]. Increased insulin levels may be associated with increased activation of the sympathetic nervous system (SNS), renin-angiotensin-aldosterone system (RAAS) activity, and variation in BP levels[13]. However, the relationships between the TyG and HRR and circadian BP patterns in normoglycemic primary hypertensive patients (NPHT) have not yet been investigated.

This study evaluated the relationships between the TyG index and cardiac autonomic function as assessed by HRR indices according to the circadian rhythm types of BP in NPHT.

Materials and Methods

A total of 1,264 patients with 24-h ambulatory BP monitoring (ABPM) in the Cardiology Clinic from 01.2019 to 01.2020 were assessed retrospectively. The study was designed considering the revised Declaration of Helsinki (2013, Brazil), following all relevant ethics protocols, and was accepted by the Ankara City Hospital Clinical Research Ethics Committee (date: 11.2022, decision no: E1-22-3054). Because of the retrospective design, the waiver of informed approval was deemed appropriate by the ethics committee that approved the study. Based on a previous study, we determined the effect size of the TyG index as 0.5 between the NDP and dipper pattern (DP)[14]. Accordingly, the sample dimension was calculated to be at least 172 with an effect size of 0.5, 90% power, and %5 alpha error probability.

Inclusion criteria were newly diagnosed NPHT patients with complete ABPM and treadmill exercise test data and no comorbidities. Exclusion criteria were previously documented hypertension, diabetes mellitus, rheumatic diseases, documented coronary artery disease, malignancy, active or chronic infection, acute or chronic kidney disease, peripheral artery disease, presence of nephrotic proteinuria, cerebrovascular disease, heart failure, liver diseases, use of antioxidants and lipid-lowering drugs, congenital or acquired valve disease, and

thyroid disease. After the exclusion process, 254 newly diagnosed primary hypertensive patients were included in the study.

All patients' clinical, ABPM, and treadmill exercise test data were obtained from the hospital's electronic information system or patient files.

Laboratory Measurements

Blood samples were drawn in the morning hours after all patients had fasted overnight. Complete blood counts were evaluated with a Sysmex XE 2100 hematology analyzer (Roche Diagnostic Corp., USA) using blood samples obtained. A photometric method was applied for assessing hemoglobin levels, an impedance method for thrombocytes and erythrocytes, and optic laser scattering for leukocytes. Lipid panels were evaluated using a Beckman Coulter LH 780 device (Beckman Coulter, Ireland). An enzymatic colorimetric method was applied for assessing lipid level measurements. The Friedewald formula was used in calculating the levels of low-density lipoprotein (LDL). Glucose was assessed with ultraviolet hexokinase using a Beckman Coulter AU 5800 autoanalyzer (Beckman Coulter, USA), and C-reactive protein (CRP) was assessed immunoturbidimetrically. The TyG index was calculated with the following formula: TyG = ln [Fasting triglyceride (mg/dL) x Fasting glucose (mg/dL)] / 2.

In-Office Blood Pressure Measurements

All participants rested for 5 min for BP measurements after admission to the hospital. Their BP levels were subsequently measured 3 times at 5-min intervals via an Omron M3 sphygmomanometer (Omron Healthcare, Japan). All measurements were averaged.

Ambulatory Blood Pressure Monitorization

A WatchBP device (Microlife WatchBP AG, Switzerland) was used to assess 24-h ABPM. Data from the first hour of monitoring were excluded from the analysis. The BP readings were automatically recorded at 15-min intervals for 24 h. Records were included in the analysis if greater than 85% of the raw records were valid. The pure reduction and percentage reduction in systolic BP for the night-time to day-time period were assessed. Bedtime was determined from the patients' diaries documenting the time of going to bed and getting up. The night-time BP levels following bed-time were evaluated from the ABPM records. Mean BP levels for the remainder of the h were evaluated as day-time BP. Diastolic BP plus 1/3 of the pulse pressure was assessed as mean BP. The percentage (%) decline in night-time BP was determined with the following formula over the averages: (daytime BP - night-time BP / daytime BP × 100). NDP was defined as the decline of night-time BP levels by <10% than day-time BP levels, while a >10% decline was defined as the DP.

The definition of hypertension is based on the European Society of Cardiology and the European Society of Hypertension.

Treadmill Exercise Testing

To investigate the BP response to exercise in all participants, a treadmill exercise test was applied. It was aimed to obtain the age-adjusted maximal HR through the modified Bruce protocol. All participants achieved 85% of their age-estimated maximum HR. Data from 12-lead ECG with the Mason-Likar modification were logged at 25 mm/s paper speed. All patients who reached the highest workload experienced at least 3 min recovery deprived cool-down. Metabolic equivalents during peak exercise were assessed as exercise capacity. Systolic and diastolic BP levels recorded at the point of maximum exercise were defined as SBPme and DBPme, respectively. HRR indices were assessed by subtracting the HRs in the first 3 min of recovery from the maximal HR during exercise. Accordingly, HRR1 for the 1st minute, HRR2 for the 2nd minute, and HRR3 for the 3rd minute were calculated.

Transthoracic Echocardiographic Examination

Echocardiographic measurements were assessed using a Vivid 7 Dimension CV Ultrasound System (General Electric Vingmed, Norway) by an experienced cardiologist. The left ventricular ejection fraction was evaluated via the modified Simpson method.

Statistical Analysis

IBM SPSS Statistics for Windows 20.0 (IBM Corp., USA) was used in the analysis of all data obtained in this study. Considering the results of the Kolmogorov-Smirnov test, numerical data with a normal distribution were identified and presented as mean ± standard deviation, while data found to have non-normal distribution were presented as median values with interquartile ranges. The Mann-Whitney U test and Student's t-test were used when comparing two groups of data with a normal distribution. Categorical variables were assessed with numbers with percentages (%), and Fisher's exact and chi-square tests were used in drawing comparisons between these groups of data. Relationships between numerical variables were assessed with Pearson correlation analysis. Multivariate linear regression analysis was assessed to identify any effects of the considered variables on decreases in the values of night-time BP (%). Values of $p<0.05$ were acceded as statistically significant.

Results

The mean age of the hypertension patients was 56.6±14.6 years and the majority of them were male (62.6%). Baseline characteristics are presented in Table 1. NDP was detected in 52.4% (n=133) of the patients. Demographic findings did not show significant differences between the NDP and DP groups. Mean neutrophil level (5.3±2.1 vs. 4.6±1.5 x10^3/µL, p=0.003), mean monocyte level (0.7±0.2 vs. 0.6±0.2 x10^3/μL, p=0.009) were higher in the NDP group than the DP group, while median lymphocyte level (2.4 vs. 2.5 x10^3/µL, p=0.046) was lower. The mean TyG index was higher in the NDP group than in the DP group (8.9±0.5 vs. 8.5±0.6, p<0.001). From echocardiographic findings, the mean left ventricular end-diastolic diameter (46.5±3.5 vs. 45.6±3.1 mm, p=0.032) and mean septum wall thickness (13.2±2.5 vs. 12.5±2.3 mm, p=0.021) were higher in the NDP group.

While the mean decline in night-time BP was 7.1%±2.5 in the NDP group, it was 15.4%±4.6 in the DP group (Table 2). Mean HRR1 (26.8±8.7 vs. 31.7±9.6 bpm, p<0.001) and mean HRR2 values were lower in the NDP group than the DP group. No correlation was found with NDP in terms of other parameters of the treadmill exercise test (Table 2).

A positive correlation was found between the TyG index and 24-hour systolic BP (r=0.418; p<0.001) and diastolic BP levels (r=0.412; p<0.001). A negative correlation was found between HRR1 values and the TyG index (r=-0.316; p<0.001) and the decline in night-time BP levels (r=-0.328; p<0.001) (Figure 1). Demographic and clinical

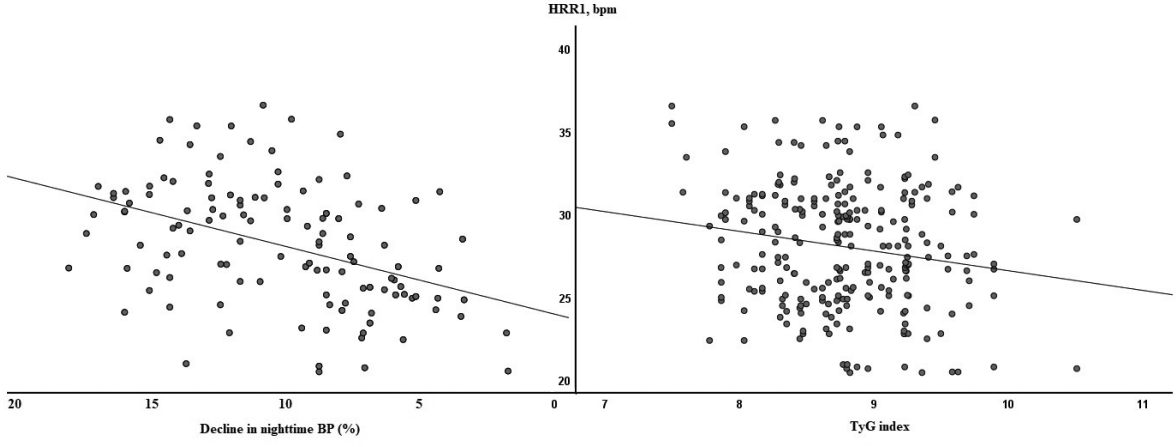

Figure 1. Relationship between HRR1 and TyG index and decline in night-time BP (%)
TyG: Triglyceride-glucose, BP: Blood pressur

parameters associated with decline in night-time BP (%) in Table 3 were included in the multivariable regression model. TyG index (β ± SE=-0.55±0.20, p<0.001), HRR1 (β ± SE=0.58±0.14, p<0.001) and DBPme (β ± SE=0.17±0.07, p=0.024) levels were determined as independent predictors of decline in night-time BP levels (%) (Table 4). Accordingly, it was determined that a 1-unit increase in the TyG index decreased the decline in night-time BP (%) levels by 0.55 folds, regardless of other risk factors. It was determined that 1 bpm increase in HRR1 level increased the decline in night-time BP (%) levels by 0.58 folds, regardless of other risk factors.

Table 1. Demographic characteristics and clinical parameters in normoglycemic primary hypertensive patients.

Variables	All population n=254	Non-dipper n=133	Dipper n=121	p-value
Demographic findings				
Age, years	56.6±14.6	55.5±15.8	57.7±13.1	0.218
Gender, n (%)				
Male	159 (62.6)	86 (64.7)	73 (60.3)	0.476
Female	95 (37.4)	47 (35.3)	48 (39.7)	
BMI, kg/m^2	24.4±3.6	24.6±3.5	24.2±3.8	0.328
Smoking, n (%)	109 (42.9)	60 (45.1)	49 (40.5)	0.458
Baseline HR, bpm	81.7±13.5	81.2±11.2	82.4±15.5	0.477
In-office SBP, mmHg	149.4±11.5	150.1±12.4	148.7±13.1	0.383
In-office DBP, mmHg	91.8±10.5	92.1±10.7	91.5±10.2	0.648
Laboratory findings				
Hemoglobin, g/dL	14.5±1.7	14.6±1.7	14.4±1.8	0.102
Glucose, mg/dL	87.3±7.4	87.4±5.5	87.1±9.1	0.688
WBC, x10^3/μL	8.4±2.3	8.6±2.4	8.2±2.1	0.135
Platelet, x10^3/μL	273.6±57.4	273.9±53.7	273.2±61.5	0.915
Neutrophil, x10^3/μL	5.0±1.8	5.3±2.1	4.6±1.5	0.003
Lymphocytes, x10^3/μL	2.5 (1.9-3.2)	2.4 (1.9-3.0)	2.6 (2.1-3.4)	0.046
Monocytes, x10^3/μL	0.7±0.2	0.7±0.2	0.6±0.2	0.009
Cholesterol, mg/dL	210.4±40.3	214.2±36.5	204.1±43.1	0.044
Triglyceride, mg/dL	140 (97-222)	158 (116-235)	127 (90-190)	0.036
HDL, mg/dL	49.3±12.2	47.4±11.5	51.2±12.7	0.016
LDL, mg/dL	123.8±39.1	118.9±33.8	129.2±43.6	0.036
Creatinine, mg/dL	0.8±0.2	0.8±0.2	0.8±0.1	0.245
CRP, mg/L	2.4 (1.3-5.2)	2.7 (1.7-5.8)	2.2 (1.2-4.3)	0.547
TyG index	8.7±0.5	8.9±0.5	8.5±0.6	<0.001
Echocardiographic findings				
Ejection fraction, %	64.2±5.3	63.8±5.2	64.5±5.4	0.294
Left atrium diameter, mm	34.7±3.6	34.6±3.9	34.9±3.3	0.581
LVEDD, mm	46.0±3.2	46.5±3.5	45.6±3.1	0.032
LVESD, mm	30.9±3.2	31.1±3.2	30.8±3.3	0.463
SWT, mm	12.8±2.3	13.2±2.5	12.5±2.3	0.021
PWT, mm	13.1±2.1	13.3±2.4	12.9±2.0	0.153

Data shown as mean±standard deviation or median (IQR) or number (percentage)
BMI: Body mass index, CRP: C-reactive protein, DBP: Diastolic blood pressure, HDL: High density lipoprotein, HR: Heart rate, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, SBP: Systolic blood pressure, SWT: Septum wall thickness, PWT: Posterior wall thickness, WBC: Leukocytes

Table 2. Ambulatory blood pressure monitorization and treadmill exercise test findings

Variables	All population n=254	Non-dipper n=133	Dipper n=121	p-value
ABPM findings				
24 hours				
SBP, mmHg	149.1±11.5	152.1±11.7	145.7±10.2	<0.001
DBP, mmHg	90.7±10.3	90.9±10.0	90.4±10.7	0.712
Day-time				
SBP, mmHg	152.0±11.6	153.1±11.7	150.8±11.3	0.109
DBP, mmHg	93.6±11.1	92.4±10.3	94.8±11.8	0.079
Night-time				
SBP, mmHg	138.6±16.5	148.9±13.7	127.3±11	<0.001
DBP, mmHg	81.2±11.8	86.4±11.0	75.5±9.8	<0.001
Decline in night-time BP, %	11.4±3.4	7.1±2.5	15.4±4.6	<0.001
Treadmill exercise test				
Duration of exercise test, min	9.1±2.3	9.3±2.4	9.0±2.1	0.292
Peak exercise capacity, METs	13.0±2.4	13.2±2.5	12.8±2.3	0.187
Maximal HR, bpm	164.5±11.2	163.9±10.4	165.1±11.8	0.390
SBPme, mm High	144.1±16.3	148.2±16.9	143.8±15.7	0.904
DBPme, mm High	96.7±12.8	98.6±12.5	94.2±13.4	0.873
HRR1, bpm	29.4±9.3	26.8±8.7	31.7±9.6	<0.001
HRR2, bpm	48.1±10.2	46.6±11.5	49.4±10.3	0.043
HRR3, bpm	63.1±14.5	62.4±15.1	63.8±13.8	0.379

Data shown as mean±standard deviation
BP: Blood pressure, DBP: Diastolic blood pressure, DBPme: Diastolic blood pressure at maximum exercise, HR: Heart rate, HRR: Heart rate recovery, METs: Metabolic equivalent levels, SBP: Systolic blood pressure, SBPme: Systolic blood pressure at maximum exercise

Discussion

To the best of our knowledge, this study is the first to report the relationship between HRR and the TyG index in terms of decline in night-time BP in NPHT patients. The results of this study showed that a high TyG index, reflecting IR, was negatively associated with HRR1 after the exercise stress test and declined in night-time BP. The TyG index was higher in the NDP group compared with the DP group, and it was an independent predictor of decline in night-time BP.

IR and impaired lipid metabolism make an important contribution to the development of hypertension[12]. There are a number of mechanisms suggested in the relationship between insulin or IR and increased BP levels. These include the activation of the SNS and RAAS with increased insulin levels, increased sodium reabsorption from the renal tubules due to hyperinsulinemia, and higher extracellular osmotic pressure than intracellular osmotic pressure due to hyperglycemia[15,16]. The TyG index showed superior diagnostic performance compared with the homeostasis model assessment of insulin resistance (HOMA-IR) in evaluating IR[17]. Increasing evidence suggests that the TyG index may be a valid population-based screening tool for hypertensive patients. A population-based study with 9 years of follow-up showed that increased TyG index was associated with cases of newly diagnosed hypertension[18]. In the current study, there was a positive correlation between the TyG index and BP levels. The above-mentioned mechanisms are also implicated in the NDP pattern of circadian BP[19,20]. Therefore, the TyG index may be an important screening tool for circadian BP variations in hypertensive patients.

Variations in circadian BP can be assessed by 24 h ABPM. It has been shown that BP changes occurring

Table 3. Parameters associated with the decline in night-time BP (%)

Variables	Decline in night-time BP (%)	
	r	p
Age	-0.088	0.160
Gender	0.024	0.708
BMI	-0.104	0.309
Smoking	0.090	0.153
Hemoglobin	0.122	0.648
Glucose	-0.288	0.018
WBC	-0.201	0.168
Platelet	-0.148	0.319
Neutrophil	-0.080	0.205
Lymphocyte	0.245	0.041
Monocyte	-0.064	0.310
Cholesterol	-0.231	0.047
Triglyceride	-0.282	0.026
HDL	0.297	0.014
LDL	-0.247	0.046
Creatinine	0.139	0.227
CRP	-0.198	0.105
TyG index	-0.328	<0.001
Basal HR	0.163	0.257
Ejection fraction	0.208	0.092
Left atrium diameter	0.119	0.259
LVEDD	-0.278	0.035
LVESD	-0.198	0.218
SWT	-0.289	0.029
PWT	-0.203	0.147
Duration of exercise test	0.118	0.358
Peak exercise capacity	0.168	0.269
Maximal HR	0.154	0.213
SBPme	0.302	<0.001
DBPme	0.311	<0.001
HRR1	0.468	<0.001
HRR2	0.279	0.035
HRR3	0.166	0.267

BMI: Body mass index, CRP: C-reactive protein, DBP: Diastolic blood pressure, DBPme: Diastolic blood pressure at maximum exercise, HDL: High density lipoprotein, HR: Heart rate, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, SBP: Systolic blood pressure, SBPme: Systolic blood pressure at maximum exercise, SWT: Septum wall thickness, PWT: Posterior wall thickness, WBC: Leukocytes

during the day can trigger CV events such as cardiac arrest, myocardial infarction, and cerebrovascular events such as hemorrhagic and ischemic stroke[21]. The NDP was found to carry higher risks of CV and cerebrovascular complications than the DP[22]. Previous studies have reported that patients with metabolic syndrome or diabetes are more prone to the NDP[23,24]. It has also been shown that IR, as assessed by HOMA-IR, is more prevalent in patients with NDP[25]. In a study of newly diagnosed hypertensive patients, TyG index and HOMA-IR levels were higher in the NDP group, and the TyG index showed a better diagnostic performance than HOMA-IR in predicting the NDP[14]. TyG index were independent predictors of declines in night-time BP and were higher in patients with NDP.

Increased insulin levels or IR plays a role in the activation of the SNS[16]. Changes in HRR levels within the first few minutes after cessation of physical exercise reflect the balance between parasympathetic and sympathetic activation[26,27]. A weakened HRR indicates a predominance of sympathetic activity and impaired parasympathetic activity. Thus, it reflects autonomic nervous system dysfunction. The finding of a negative correlation between HRR1 and the TyG index supports the role of IR in sympathetic and parasympathetic activation. Increased sympathetic activity, increased hemodynamic stress, and CV workload are associated with cardiac morbidity and mortality[28].

Therefore, a weakened HRR is associated with CV dysfunction and events[8,29]. A prospective study involving a 4-year follow-up of healthy subjects reported that reduced HRR2 was associated with the development of hypertension[11]. NPHT patients with NDP were found to exhibit lower HRR1 levels. A study of primary hypertension patients reported that HRR1 levels were lower in patients with NDP, and there was a positive correlation between HRR1 and decline in night-time BP percentage[10]. The study of hypertensive patients conducted by Kim et al.[30] reported similar results. In another study, it was shown that HRR values were lower

Table 4. Independent predictors of the decline in night-time BP (%)

Variables	Univariable model			Multivariable model		
	β±SE	95% CI Lower, Upper	p-value	β±SE	95% CI Lower, Upper	p-value
Glucose	-0.15±0.07	-0.30; -0.03	0.018	-	-	-
Lymphocyte	0.18±0.07	0.0, 0.32	0.041	-	-	-
Cholesterol	-0.24±0.11	-0.47; -0.02	0.047	-	-	-
Triglyceride	-0.18±0.06	-0.30; -0.06	0.026	-	-	-
HDL	-0.03±0.01	-0.0, 0.02	0.014	-	-	-
LDL	0.05±0.02	-0.10; -0.01	0.046	-	-	-
TyG index	-0.51±0.17	-0.85; -0.17	<0.001	-0.55 ± 0.20	-0.95; -0.15	<0.001
LVEDD	-0.37±0.16	-0.69; -0.05	0.035	-	-	-
SWT	-0.40±0.18	-0.76; -0.04	0.029	-	-	-
SBPme	0.15±0.04	0.0, 0.30	<0.001	-	-	-
DBPme	0.16±0.05	0.0, 0.32	<0.001	0.17 ± 0.07	0.0, 0.31	0.024
HRR1	0.57±0.09	0.4, 0.75	<0.001	0.58 ± 0.14	0.3, 0.86	<0.001
HRR2	0.24±0.11	0.0, 0.46	0.035	-	-	-
				Adjusted R^2=0.285; p<0.001		

β: Regression coefficient, CI: Confidence interval, DBPme: Diastolic blood pressure at maximum exercise, HDL: High density lipoprotein, HR: Heart rate, LVEDD: Left ventricular end-diastolic diameter, LVESD: Left ventricular end-systolic diameter, SBPme: Systolic blood pressure at maximum exercise, SE: Standard error, SWT: Septum wall thickness, PWT: Posterior wall thickness

in hypertensive patients with uncontrolled BP values than hypertensive patients with controlled BP value[31]. In this study, HRR1 and HRR2 were associated with a decline in night-time BP percentage, whereas HRR3 was not. The reactivation of the parasympathetic nervous system exerts a stronger effect in the first 60 seconds after the end of exercise and mediates the response to activity in arterial baroreceptors[32]. Catecholamines, which play an important role in the autonomic nervous system, reach peak levels during peak exercise and may not return to normal levels until about 90 seconds of recovery[33]. These findings observed within the first 2 min of the recovery phase may be associated with a better reflection of autonomic nervous system dysfunction in this phase. This may have caused a meaningless relationship between the 3rd minute of the recovery (HRR3) and the decline in night-time BP percentage. On the other hand, decreased HRR1 levels were the strongest predictors of decline in night-time BP percentage. These results demonstrate that the SNS plays a role in unsuccessful night-time BP reduction. Therefore, the TyG index may be an effective predictor of variations in the circadian BP and autonomic nervous system beyond CV events[34].

Study Limitations

This study has some important limitations. Initially, it had a retrospective design and the sample dimension was relatively small. Second, the relationship among the TyG index and NDP and HRR has been linked to the autonomic nervous system. To fully clarify that relationship, evaluation of sympathetic activation with gold-standard measurement methods such as cardiac metaiodobenzylguanidine scintigraphy with ^{123}iodine labeling would have added more power to the study. Finally, the relationship among the TyG index, HRR, and target organ damage resulting from hypertension could not be evaluated, because the retrospective design.

Conclusion

Elevated TyG index was associated with delayed HRR and NDP after exercise stress tests. Increased TyG index

and decreased HRR1 levels were important predictors of decline in night-time BP percentage. NPHT patients may be at risk of autonomic dysfunction due to increased IR.

Ethics

Ethics Committee Approval: Ethics Committee Approval was obtained from the Ankara City Hospital Clinical Research Ethics Committee (date: 11.2022, decision no: E1-22-3054).

Informed Consent: The study was designed as a retrospective.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Concept: Doğanay B, Design: Doğanay B, Data Collection and/or Processing: Doğanay B, Başar V, Analysis and/or Interpretation: Doğanay B, Başar V, Literature Search: Doğanay B, Başar V, Writing: Doğanay B, Başar V.

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