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Original Article

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Noninvasive Subclinical Atherosclerosis Indicator in a New Diagnosis of Primary Hypertension: Blood Pressure Index

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Abstract

Objective: Multiple parameters can be used for determination of hypertension-related end-organ damage (EOD) in the cardiovascular system. Carotid intima-media thickness (CIMT), left ventricular mass index (LVMI), urinary albumin excretion (UAE), and retinopathy are the frequently used parameters for hypertension-related EOD. We investigated the importance of the blood pressure (BP) index, which we defined as the ratio of systolic BP (SBP) to diastolic BP (DBP) in determining subclinical atherosclerosis.

Materials and Methods: A total of 205 patients above 18 years of age who were being followed up with a new diagnosis of essential hypertension were included in this study. The LVMI, CIMT, and UAE levels of the patients were recorded from the patient files.

Results: The 24-h BP index and the nighttime and daytime BP indices were determined to be higher in the EOD-positive group than in the EOD-negative group. The 24-h BP index and the nighttime and daytime BP indices were determined to be associated with UAE, LVMI, and CIMT. In the regression analysis performed, the 24-h BP index was determined to be a risk factor associated with EOD, similar to other findings of subclinical atherosclerosis.

Conclusion: We think that the BP index may be a favorable indicator in determination of primary-hypertension-related EOD.

Keywords: Atherosclerosis, blood pressure, hypertension

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INTRODUCTION

Primary hypertension is among the group of important diseases that are included in the etiopathogenesis of major cardiovascular complications (syncope, acute coronary syndrome, and peripheral artery disease) worldwide (1-4). High blood pressure (BP) resulting from increased cardiac output and peripheral vascular resistance causes cardiac and vascular remodeling, initially causing subclinical cardiovascular diseases and subsequently organ dysfunction (5). Thus, the diagnosis of hypertension and hypertension-associated complications in the early phase and taking necessary measures are of vital importance in preventing potential cardiovascular complications (6). Hypertension causes endothelial dysfunction, leading to subclinical atherosclerosis in central and peripheral vascular structures in the early phase (7). In clinical practice, the process of atherosclerosis can be decelerated when end-organ damage (EOD) is diagnosed in the subclinical phase and necessary measures are taken in hypertensive cases. Many parameters can be used for determination of EOD in the cardiovascular system. Carotid intima-media thickness (CIMT), flow-dependent dilation measured from the brachial artery, increase in left ventricular mass index (LVMI), urinary albumin excretion (UAE), and retinopathy are frequently used parameters for EOD (8).

It might not be feasible to test these indicators of subclinical atherosclerosis in all the hospitals and clinical settings because it is not cost effective and there is a lack of appropriate medical resources (devices, experts, etc.). Therefore, the possible usage of simple, cost-effec-

tive, and accessible techniques by which these parameters can be evaluated in patients who are being followed with a diagnosis of hypertension is of vital clinical importance.

We assumed that the BP index, which we defined as the ratio of systolic BP (SBP) to diastolic BP (DBP), may be a favorable indicator for subclinical atherosclerosis. Therefore, we aimed to investigate the association of the BP index with CIMT, LVMI, and UAE, which are the indicators of subclinical atherosclerosis in patients with hypertension.

MATERIALS AND METHODS

Patient Selection

This was a retrospective study conducted at Ankara Numune Training and Research Hospital between March 2017 and July 2017.

A total of 205 patients above 18 years of age who were being followed up with a diagnosis of essential hypertension in the Department of Internal Diseases between 2014 and 2016 were included in the study. Hypertension was diagnosed for the first time in our Department of Internal Diseases as recorded in patient files.

Patients with secondary hypertension, diabetes mellitus, obesity, acute/chronic kidney disease, primary nephrotic syndrome, documented coronary artery disease, heart failure, peripheral artery disease, cerebrovascular disease, malignancy, liver diseases, rheumatic diseases, active or chronic infections, usage of any antioxidant substances or lipid-lowering medications, tobacco or alcohol consumption, or usage of vitamin supplementation were excluded from the study.

Informed consent was obtained from the patients who participated in this study. The study was planned in accordance with the Declaration of Helsinki and the Regulation of Patient's Rights, and it was approved by the Clinical Research Ethics Board of Ankara Numune Training and Research Hospital (Approval Date: February 2, 2017; Approval Number: 1253-2017).

Parameters Investigated in the Study

Laboratory parameters were recorded from the patients' electronic files. Attention was paid to all laboratory parameters obtained from the blood samples collected in the morning on an empty stomach. The devices and methods used in the Department of Biochemistry of our hospital for the measurement of these laboratory parameters are specified below.

Glucose was measured by the ultraviolet hexokinase method on a Beckman Coulter AU 5800 autoanalyzer (Beckman Coulter Inc.; Brea, CA, USA). Triglycerides and total cholesterol were measured by an enzymatic colorimetric method, high-density lipoprotein cholesterol by a homogeneous enzymatic colorimetric method, and 24-h urinary protein and microalbumin by a turbidimetric method on a Hitachi Modular P800 autoanalyzer (Roche Diagnostic Corp.; Indianapolis, IN, USA) (9). Erythrocytes and thrombocytes were measured by impedance (resistance) method, leukocytes by optic laser scattering (light scattering), and other hematological parameters by Sysmex XE 2100 hematology analyzer (Roche Diagnostic Corp; Indianapolis, IN, USA). Hemoglobin was measured photometrically.

UAE of 30-300 mg, LVMI of >95 in women and >115 in men, and CIMT of >0.9 were considered as evidence of EOD.

Ambulatory Blood Pressure Measurement

A WatchBP 03 ABPM device (Microlife WatchBP AG; Widnau, Switzerland) was applied to all patients for 24-h ambulatory BP measurement (ABPM). The ABPM device was set for 30-min interval measurements. The patients were allowed to continue their daily activities. The information necessary for the ABPM device to make accurate measurements was provided to participants. As a result, the 24-h SBP, 24-h DBP, nighttime SBP, nighttime DBP, daytime SBP, and daytime DBP measurements were obtained. At the end of the 24-h measurements, making more than 70% of the measurements in total was considered to be significant for ABPM.

Echocardiography

Echocardiography procedures were performed with an echocardiography device (2.5-MHz transducer, Vivid 7, GE-Vingmed Ultrasound AS, Horten, Norway) by a cardiologist who was blinded to the study. Complete 2D (dimension), color, beat and continuous wave Doppler examinations were performed in accordance with standard techniques. For obtaining M-mode measurements of left atrial size, left ventricular end-diastolic septum and posterior wall thickness, and left ventricular systolic and diastolic dimensions, parasternal long axis images were used. LVMI was calculated using 2D echocardiographic measurements with the Devereux formula, which is defined as Left Ventriculer Mass=1.04×[(Interventricular Septum Thickness + Posterior Wall Thickness + Left Ventricular Internal Diameter in Diastole)^3 - (Left Ventricular Internal Diameter in Diastole)^3] - 13.6, and it was indexed to body surface area (10). Mean LVMI of >100 g/m^2 was considered to be significant for left ventricular hypertrophy.

Carotid Doppler Ultrasonography

CIMT was measured with the patient in the supine position and both hands under his or her head. The measurements were

Main Points

- The 24-h BP index and the nighttime and daytime BP indices were determined to be associated with urinary albumin excretion, left ventricular mass index and Carotid intima-media thickness.
- The 24-h BP index was determined to be a risk factor associated with end-organ damage.
- It was determined that an increase of 1% in 24-h BP index increased the risk of EOD 1.47-fold.

performed using a high-resolution B-mode device (Logic 7, GE Med. Inc.; Bloomington, IL, USA) by a radiologist who had no knowledge about the clinical statuses of the patients. In these measurements, the right and left main carotid arteries were evaluated with an automated system by a linear probe. The measurements were performed from 3 points: right and left main carotid arteries, bifurcation, and first 2-cm part of internal carotid arteries. Longitudinal measurements were performed from the distances defined between vascular lumen echogenicity and media adventitia echogenicity. CIMT was calculated by taking the average of 3 measurements made for both the carotid arteries.

Statistical Analysis

Statistical evaluation was performed using the Statistical Package for the Social Sciences 20 for Windows (IBM Corp.; Armonk, NY, USA). Normal distribution of the data was evaluated using the Kolmogorov-Smirnov test. Numerical variables that exhibited normal distribution were given as mean±standard deviation, and those that did not exhibit normal distribution were represented as median (min-max). Categorical variables were presented as numbers and percentages. For determination of factors associated with two-category risk groups, the t-test (for numerical variables exhibiting normal distribution) and Mann-Whitney U test (for numerical variables without normal distribution) were used for independent samples. For comparison of categorical data, the chi-squared test and Fisher's exact chi-squared test were used. The association between numerical variables was evaluated with the Pearson and Spearman correlation analysis. Predictors that were found to be related to EOD ($p \leq 0.25$) were then entered into a multivariate logistic regression model, using stepwise backward selection. The cut-off values of independent numerical predictors were evaluated by receiver operating characteristic curve analysis with the Youden index method. Values of $p<0.05$ were considered to be statistically significant.

RESULTS

The study population included 205 patients with hypertension, including 144 women and 61 men. The UAE levels of the patients were between 1.1 and 300 mg/day with a median of 10.1 mg/day, and UAE was 30 mg/day or above in 18.5% of the patients. The mean LVMI level of the patients was 88±22 g/m^2, the percentage of patients with LVMI of >95 g/m^2 was 23.9% of the whole population among women, and the percentage of patients with LVMI of >115 g/m^2 was 6.8% of the whole population among men. The mean CIMT level was 0.8±0.2 mm, and the percentage of patients with CIMT of >0.9 mm was 30.2%. The percentage of patients with determined EOD was 38.5% (n=79).

The distribution of demographic, clinical, and laboratory findings according to presence of EOD is shown in Table 1. In the EOD-positive group, the median UAE level (23 versus 7.9; $p<0.001$), mean LVMI level (105.1±22.4 versus 77.3±13.2; $p<0.001$), and mean CIMT (1.0±0.2 versus 0.7±0.1; $p<0.001$) lev-

Table 1. Distribution of demographic, clinical, and laboratory findings according to EOD

Variables	EOD (+) (n=79)	EOD (-) (n=126)	p
Demographic findings			
Gender, n (%)			
Female	52 (65.8)	92 (73.0)	0.277
Male	27 (34.2)	34 (27.0)	
Age (years)	54.9±12.3	52.0±12.3	0.106
BMI (kg/m^2)	29.6±4.7	29.9±5.0	0.608
Laboratory findings			
Hemoglobin (g/dL)	14.0±1.6	13.8±1.7	0.364
Platelet (×10^3 µL)	271.8±79.7	286.5±69.6	0.169
WBC count (µL)	6,971.7±2,268.9	7,451.5±2,305.2	0.147
Total cholesterol (mg/dL)	201.3±42.5	206.1±49.7	0.481
LDL cholesterol (mg/dL)	117.9±37.4	124.3±36.1	0.224
HDL cholesterol (mg/dL)	50.4±16.2	49.5±12.3	0.676
Triglycerides (mg/dL)	142 (55-735)	138 (34-1951)	0.592
C-reactive protein (mg/L)	3.2 (0.4-92)	3.2 (0.3-94.9)	0.560
UAE (mg/day)	23 (1.4-300)	7.9 (1.1-29)	<0.001*
LVMI (g/m^2)	105.1±22.4	77.3±13.2	<0.001*
CIMT (mm)	1.0±0.2	0.7±0.1	<0.001*
ABPM findings			
24-h SBP (mm Hg)	151.9±16.4	142.5±11.9	<0.001*
24-h DBP (mm Hg)	94.8±8.4	90.5±8.5	<0.001*
24-h BP index	1.7±0.1	1.5±0.1	<0.001*
Daytime SBP (mm Hg)	154.7±15.5	146.6±12.9	<0.001*
Daytime DBP (mm Hg)	99.4±8.4	93.5±9.3	<0.001*
Daytime BP index	1.7±0.1	1.5±0.1	<0.001*
Nighttime SBP (mm Hg)	148.1±18.8	135.6±12.9	<0.001*
Nighttime DBP (mm Hg)	83.2±9.4	85.6±9.3	0.075
Nighttime BP index	1.8±0.1	1.6±0.1	<0.001*

*p<0.05 is statistically significant
Categorical variables are presented as number (percentage), and numerical variables are expressed as mean±standard deviation
EOD: end-organ damage; BMI: body-mass index; WBC: white blood cell; LDL: low-density lipoprotein; HDL: high-density lipoprotein; UAE: urine albumin excretion; LVMI: left ventricular mass index; CIMT: carotid intima-media thickness; ABPM: ambulatory blood pressure monitoring; SBP: systolic blood pressure; DBP: diastolic blood pressure; BP: blood pressure

Table 2. Clinical and laboratory findings associated with subclinical atherosclerosis markers in patients with primary hypertension

	UAE		LVMI		CIMT	
Variables	**r**	**p**	**r**	**p**	**r**	**p**
Age	0.077	0.276	0.143	0.142	0.084	0.237
BMI	0.057	0.433	-0.053	0.465	0.005	0.944
Hemoglobin	0.085	0.224	-0.003	0.966	0.084	0.235
Platelet	-0.044	0.530	-0.064	0.362	-0.112	0.111
WBC count	-0.025	0.719	-0.025	0.727	-0.079	0.263
Total cholesterol	0.032	0.647	-0.037	0.603	-0.030	0.672
LDL cholesterol	0.000	0.997	-0.051	0.474	-0.083	0.240
HDL cholesterol	0.064	0.368	-0.042	0.555	0.047	0.502
Triglycerides	0.018	0.804	0.029	0.677	0.011	0.879
C-reactive protein	-0.003	0.971	-0.004	0.960	0.046	0.511
UAE	-	-	0.348	<0.001*	0.380	<0.001*
LVMI	0.348	<0.001*	-	-	0.464	<0.001*
CIMT	0.380	<0.001*	0.464	<0.001*	-	-
24-h SBP	0.323	0.029*	0.324	0.026*	0.358	<0.001*
24-h DBP	0.274	0.031*	0.290	0.033*	0.326	0.027*
24-h BP index	0.353	<0.001*	0.398	<0.001*	0.607	<0.001*
Daytime SBP	0.311	0.027*	0.287	0.045*	0.329	0.031*
Daytime DBP	0.299	0.025*	0.245	0.034*	0.307	0.036*
Daytime BP index	0.347	0.002*	0.395	<0.001*	0.592	<0.001*
Nighttime SBP	0.299	0.039*	0.288	0.037*	0.362	0.011*
Nighttime DBP	0.235	0.075	0.227	0.106	0.226	0.268
Nighttime BP index	0.315	0.005*	0.358	<0.001*	0.464	<0.001*

*p<0.05 is statistically significant
UAE: urine albumin excretion; LVMI: left ventricular mass index; CIMT: carotid intima-media thickness; BMI: body-mass index; WBC: white blood cell; LDL: low-density lipoprotein; HDL: high-density lipoprotein; SBP: systolic blood pressure; DBP: diastolic blood pressure; BP: blood pressure

el were determined to be higher than those in the EOD-negative group. In the EOD-positive group, the mean 24-h SBP level (151.9±16.4 versus 142.5±11.9; p<0.001), mean 24-h DBP level (94.8±8.4 versus 90.5±8.5; p<0.001), mean daytime SBP level (154.7±15.5 versus 146.6±12.9; p<0.001), mean daytime DBP level (99.4±8.4 versus 93.5±9.3; p<0.001), and mean nighttime SBP level (148.1±18.8 versus 135.6±12.9; p<0.001) were determined to be higher than those in the EOD-negative group, but mean nighttime DBP did not significantly differ (83.2±9.4 versus 85.6±9.3; p<0.068). Mean 24-h BP index, daytime BP index, and nighttime BP index were higher in the EOD-positive group than those in the EOD-negative group (p<0.001).

Table 2 shows the clinical and laboratory findings associated with indicators of subclinical atherosclerosis in patients with primary hypertension. Accordingly, a positive correlation was determined between UAE and LVMI (r=0.348; p<0.001), CIMT (r=0.380; p<0.001), 24-h SBP level (r=0.323; p=0.029), 24-h DBP level (r=0.274; p=0.031), daytime SBP level (r=0.311; p=0.027), daytime DBP level (r=0.299; p=0.025), and nighttime SBP level (r=0.299; p=0.039). A positive correlation was determined between LVMI and CIMT (r=0.464; p<0.001), 24-h SBP level (r=0.324; p=0.026), 24-h DBP level (r=0.290; p=0.033), daytime SBP level (r=0.287; p=0.024), daytime DBP level (r=0.245; p=0.034), and nighttime SBP level (r=0.288; p=0.037). A positive correlation was determined between CIMT and 24-h SBP level (r=0.358; p<0.001), 24-h DBP level (r=0.326; p=0.027), daytime SBP level (r=0.329; p=0.031), daytime DBP level (r=0.307; p=0.036), and nighttime SBP level (r=0.362; p=0.011).

Table 3. Independent markers for end-organ damage

Possible risk factors	Univariate			Multivariate		
	OR	95% CI	p	OR	95% CI	p
Age	1.01	0.98-1.04	0.106	-	-	-
BMI	0.97	0.91-1.03	0.608	-	-	-
Platelet	1	0.95-1.06	0.169	-	-	-
WBC	1	0.97-1.05	0.147	-	-	-
24-h SBP	1.05	1.02-1.07	<0.001*	-	-	-
24-h DBP	0.93	0.89-0.96	<0.001*	-	-	-
24-h BP index	1.45	1.29-1.62	<0.001*	1.47	1.30-1.65	<0.001*
Daytime SBP	1.04	1.02-1.06	<0.001*	-	-	-
Daytime DBP	0.93	0.89-0.96	<0.001*	-	-	-
Daytime BP index	1.24	1.16-1.32	<0.001*	-	-	-
Nighttime SBP	1.05	1.03-1.07	<0.001*	-	-	-
Nighttime DBP	0.98	0.940-1.02	0.075	-	-	-
Nighttime BP index	1.21	1.15-1.27	<0.001*	-	-	-
				Nagelkerke R^2=0.774; p<0.001		

*p<0.05 is statistically significant
Demographic, clinical, and laboratory parameters shown in Table 1 were evaluated in the univariate regression model with p<0.25
OR: odds ratio; CI: confidence interval; BMI: body-mass index; WBC: white blood cell; SBP: systolic blood pressure; DBP: diastolic blood pressure; BP: blood pressure

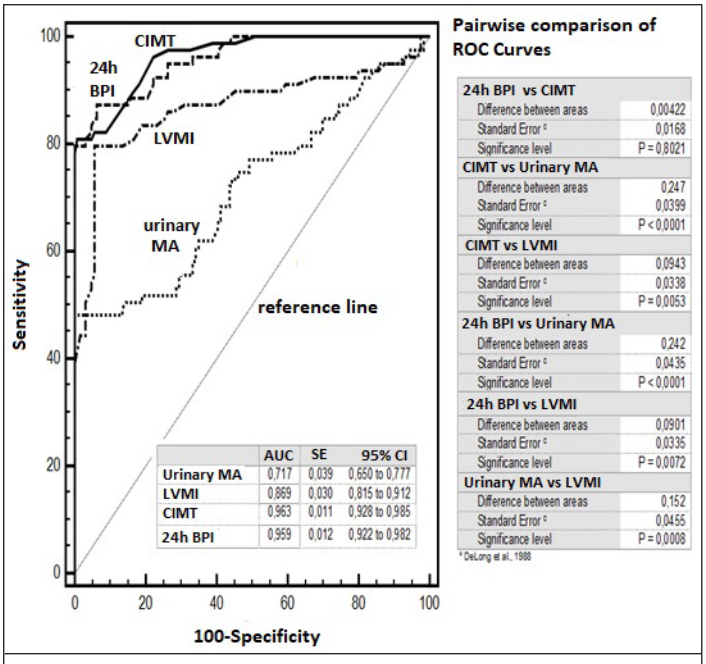

Figure 1. Diagnostic differentials of independent predictors in predicting end-organ damage

A positive correlation was determined between UAE levels and 24-h BP index (r=0.353; p<0.001), daytime BP index (r=0.347; p=0.002), and nighttime BP index (r=0.315; p=0.005). A positive correlation was determined between LVMI levels and 24-h BP index (r=0.607; p<0.001), daytime BP index (r=0.592; p<0.001), and nighttime BP index (r=0.464; p<0.001).

Variables associated with EOD are presented in Table 1. Accordingly, age, body-mass index, platelet, white blood cell, and ABPM parameters were evaluated in the univariate regression model with p≤0.25. These parameters were then entered into a multivariate logistic regression model, using stepwise backward selection. As a result of multivariate analysis, it was determined that only 24-h BP index (odds ratio=1.47; p<0.001) predicted target organ damage (Table 3). It was determined that an increase of 1% in 24-h BP index increased the risk of EOD 1.47-fold.

Values of the 24-h BP index above 1.62 were determined to predict EOD with a sensitivity of 87.3% and a specificity of 93.7% (area under the curve±standard error of mean: 0.959±0.013; p<0.001) (Figure 1).

DISCUSSION

In our study including 205 patients with primary hypertension, the 24-h BP index, nighttime BP index, and daytime BP index were determined to be associated with UAE, LVMI, and CIMT, which are all the indicators of subclinical atherosclerosis. Furthermore, among these indicators, the 24-h BP index was de-

termined to be an independent risk factor for findings of subclinical atherosclerosis. The values of the 24-h BP index and the nighttime and daytime BP indices were determined to be higher in the EOD-positive group than those in the EOD-negative group. In the regression analysis performed, the 24-h BP index was determined to be a risk factor associated with EOD, similar to other findings of subclinical atherosclerosis. When the diagnostic value of these parameters in predicting EOD was examined, it was found that 24-h BP index and CIMT had similar diagnostic value; however, these 2 parameters had a higher diagnostic value than that of UAE and LVMI. To the best of our knowledge, this is the first study investigating the role of BP index in determining subclinical atherosclerosis in patients with primary hypertension.

In our literature review, we did not encounter any study investigating the role of the BP index in a primary hypertension group. Furthermore, how this index changes in the case of hypertension-induced subclinical atherosclerosis is not clearly understood. In a study conducted by Ates et al. (11) the association of the BP index with right ventricular dysfunction was investigated in patients with acute pulmonary embolism. As is known, the initial effect of acute pulmonary embolism on cardiac function is peripheral BP alteration. In the same study, it was demonstrated that BP index was an independent parameter in indicating right ventricular dysfunction, with a negative association. In acute pulmonary embolism, the right ventricular end-diastolic pressure increases because of right ventricular dysfunction, and the interventricular septum shifts toward the left ventricle, resulting in the reduction in the left ventricular stroke volume. SBP is associated with left ventricular stroke volume and decreases with the decrease in stroke volume, and it was suggested that a greater decrease of SBP than DBP could result in the reduction of the BP index (11).

In our study, however, as an indicator of subclinical atherosclerosis, high BP index values were observed to be associated with atherosclerosis. As is known, left ventricular hypertrophy develops because of an increase in the afterload in hypertension. This condition may lead to an increase in the left ventricular contractility. Although the increase in contractility suggests that stroke volume and therefore SBP may increase, clinical experience does not support this. The reason for this is that left ventricular hypertrophy is accompanied by diastolic functional impairment and may lead to reduction of preload, which is another important determinant of stroke volume. A possible explanation for the increase in BP index may be the reduction of elasticity of the aorta owing to hypertension and an expected increase in SBP, as stipulated by the Laplace law, owing to the ineffective enlargement of the aortic wall during systole. The fact that this increase is more prominent than the increase of DBP may be explained by the high level of BP index (12, 13).

Furthermore, in our study, although positive correlations were observed between 24-h SBP, 24-h DBP, daytime SBP, daytime DBP, and nighttime SBP and LVMI, CIMT, and UAE, which are the indicators of subclinical atherosclerosis, no association with nighttime DBP was determined.

In a review of the previous studies in the literature, the associations among UAE, arterial stiffness, daytime SBP, and nighttime SBP were investigated, and nighttime SBP was determined to be more specific than daytime SBP. Nighttime SBP was demonstrated to be in closer association with cardiovascular outcomes, including development of EOD, stroke, and death, than 24-h SBP (14-16). Owing to the lack of physical and mental stress factors during sleep, nighttime BP is equal to the minimal blood pressure required for sufficient perfusion of tissues. When nighttime BP is above this basal limit, hemodynamic overload occurs, and the development of EOD may be triggered. As a result of the lack of physical and mental activities throughout the night, alterations in the BP decrease, and this can be evaluated only by 24-h ABPM. Therefore, nighttime BP is an important indicator of hypertension-related hemodynamic load (14, 16).

No study investigating the role of BP index in patients with primary hypertension has been encountered in the literature; however, the results of a few studies on pulse pressure can be explained by a pathophysiological mechanism similar to that of the BP index. In a study by Ruiz-Hurtado et al., (15) the course of daytime and nighttime SBP and DBP was examined in patients with hypertension, and the presence of increased pulse pressure was revealed. This indicates that increased pulse pressure in large-sized and small-sized arteries facilitates the damage of small-sized vessels in the brain, heart, and kidneys (15, 17). Furthermore, in many of the reviewed studies, ambulatory pulse pressure was demonstrated to be superior to daytime pulse pressure in indicating LVMI (18, 19). By definition, the BP index and pulse pressure are similar parameters. In a study by Ruiz-Hurtado et al., (15) increase in pulse pressure was determined to be associated with subclinical atherosclerosis, and this finding suggests that BP index and subclinical atherosclerosis may be associated.

In some previous studies, however, the role of the pulse pressure index (pulse pressure/SBP) in indicating EOD was investigated (20). With increasing age, SBP increases more than DBP, and this leads to an increase in the pulse pressure. An increase in the vascular stiffness because of loss of elasticity in the aorta and large-sized arteries is an important cardiovascular risk factor and is the cause of this alteration in the pulse pressure (20). Diffuse atherosclerotic lesions, the aging process of arterial walls, and increased pulse pressure because of other mechanisms are thought to be important indicators of arterial stiffness (21). In a study conducted by Verdecchia, a 24-h ambulatory pulse pressure of >53 mm Hg was determined to be associated with an increased cardiovascular risk (22). As BP exhibits variations throughout the day, pulse pressure is not stable. On the basis of this hypothesis, in a study by Ede et al., (23) the association of pulse pressure index and left ventricular diastolic

dysfunction was investigated, and it was revealed that New York Heart Association (NYHA) class 2 patients had higher pulse pressure index values than those of NYHA class 1 patients.

As primary hypertension is an atherosclerotic and chronic inflammatory disease, EOD occurs frequently (24). To investigate whether the BP index has a role in the prevention of EOD in hypertension, the disease should be diagnosed, and preventive treatments and treatments reversing the damage should be initiated while the illness is still in an asymptomatic stage. In this study, we examined the association of the indicators of primary-hypertension–induced EOD with the BP index. The BP index was determined to be higher in the group with EOD than that in the group without it. Furthermore, it was determined that the severity of EOD and the level of increase in BP index showed parallelism.

For this index to be used as an indicator of EOD in clinical practice, the BP index level should be reevaluated after preventive treatments, treatments to ameliorate or reverse the damage, and treatments to control the blood pressure are initiated. In this setting, if there is a regression of the BP index, it would be possible to conclude that the BP index may be a favorable indicator for the diagnosis and follow-up of EOD.

The retrospective cross-sectional design of this study is its greatest limitation. Not testing other indicators involved in the pathogenesis of atherosclerosis and inflammatory markers with high sensitivity that can indicate chronic inflammation is an additional limitation. Another limitation is that we included only newly diagnosed patients with hypertension and patients without comorbidities, such as diabetes mellitus and cardiovascular disease; therefore, the results of this study may not be applicable to all patients with primary hypertension.

This study was not designed to obtain objective data; however, it may give insights to clinicians for determination of the development of EOD. Randomized controlled studies are needed for the BP index to be used routinely.

CONCLUSION

In our study, the 24-h BP index, nighttime BP index, and daytime BP index were determined to be associated with UAE, LVMI, and CIMT, which are the indicators of subclinical atherosclerosis in the primary hypertension group. In light of these results, we can conclude that the BP index may be a reliable indicator of primary-hypertension-related EOD.

Ethics Committee Approval: Ethics committee approval was received for this study from the Clinical Research Ethics Board of Ankara Numune Training and Research Hospital (Approval Date: February 2, 2017; Approval Number: 1253-2017).

Informed Consent: Informed consent was obtained from the patients who participated in this study.

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