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The relationship between proteinuria and ambulatory blood pressure in hypertensive patients

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ABSTRACT

Objectives: The study aimed to investigate the relationship between proteinuria and blood pressure (BP) determined with ambulatory BP monitoring (ABPM) in patients who applied to the nephrology clinic due to hypertension.

Patients and methods: A total of 163 patients (84 males, 79 females; mean age: 55.7±16.6 years; range, 18 to 80 years) were included in the cross-sectional study between January 2022 and January 2023. The amount of proteinuria was measured from 24-h urine samples. The ABPM values were measured using noninvasive multitasking BP recorders.

Results: A total of 53.4% (n=87) of the patients had dipper, 29.4% (n=48) had non-dipper, and 17.2% (n=28) had reverse-dipper hypertension (HT). Dipper HT, albumin, and glomerular filtration rate were significantly lower in those with proteinuria compared to those without proteinuria. Age, creatinine, HT duration, 24-h, daytime, and nighttime systolic BP, nighttime diastolic BP, nighttime mean BP, non-dipper HT (all $p<0.001$), 24-h diastolic BP ($p=0.015$), daytime mean BP ($p=0.005$), and reverse-dipper HT ($p=0.001$) were significantly higher in the group with proteinuria.

Conclusion: Elevated ABPM values, non-dipper HT, and reverse-dipper HT were detected in patients who had high proteinuria. Creatinine and 24-h urine protein excretion were found to be higher in patients with non-dipper HT and reverse-dipper HT. The progression of proteinuria can be slowed down by strict BP control in hypertensive patients with proteinuria.

Keywords: Ambulatory blood pressure monitoring, hypertension, proteinuria.

Hypertension (HT) is a risk factor affecting more than one billion people worldwide and leads to high mortality, but control rates are low.[1] It is already known that effective control of blood pressure (BP) reduces cardiovascular disease and renal morbidity and mortality.

Different methods are used to monitor the BP levels of patients. These are office BP measurement, home BP measurement, and ambulatory BP monitoring (ABPM). Constanti et al.[2] reported that office BP measurement is limited to a comprehensive BP check. An important aspect of the information provided with the ABPM is the ability to measure the degree of BP variability over 24 h, which was shown to be a significant and independent risk factor for cardiovascular disease morbidity and mortality.[3] Variability in BP includes short-term and circadian components. The drop in BP over time causes large variability among individuals. Elevated BP in the early morning hours, when cardiovascular events occur most frequently, can be detected early with ABPM.[4] Microalbuminuria is among the organ damages associated with HT, and its prevalence was reported to be between 8 and 15% in the nondiabetic patient population. Determination of microalbuminuria facilitates the approach to treatment and risk assessment for hypertensive patients.[5] Patients who have HT often

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also have renal damage. This condition manifests itself as microalbuminuria. The relationship between microalbuminuria and HT is explained by endothelial dysfunction or chronic low-grade inflammation. This occurs because BP fluctuation, particularly at night, increases glomerular perfusion pressure, causes endothelial cell damage in glomerular capillaries, increases microalbuminuria, and causes progressive renal damage.[4] Microalbuminuria is also associated with chronic low-grade inflammation, which can be a cause and a consequence of endothelial dysfunction. Furthermore, endothelial dysfunction and low-grade inflammation not only cause atherothrombosis but may also be independently associated with cardiovascular disease risks. When BP fluctuates, the sympathetic nervous system is stimulated, endocrine regulation is disrupted, and renal damage is triggered.[6] Although previous studies were conducted to evaluate ABPM in hypertensive patients, this study aimed to investigate the relationship between proteinuria levels and BP fluctuations determined with ABPM.

PATIENTS AND METHODS

The cross-sectional study was conducted with a total of 163 patients (84 males, 79 females; mean age: 55.7±16.6 years; range, 18 to 80 years) who were diagnosed with HT at the Ankara Bilkent City Hospital, Department of Nephrology, between January 2022 and January 2023. Those who were over 18 years of age, without diabetes, not under treatment with steroids or other immunosuppressive drugs, and without malnutrition, active malignancy, active infection, a history of myocardial infarction, cerebrovascular disease in the last six months, unstable angina, or other major diseases were included in the study. The study protocol was approved by the Ankara City Hospital Ethics Committee (date: 09.12.2020, no: E1-20-1355). Written informed consent was obtained from all participants. The study was conducted in accordance with the principles of the Declaration of Helsinki. Venous blood samples were taken from the patients between 8:00 and 9:00 in the morning after an 8- to 10-h fast. Serum creatinine levels were analyzed with the spectrophotometric method by using the Beckman Coulter commercial kits on the Beckman Coulter AU5800 autoanalyzer (Beckman Coulter, Inc., Brea, CA, U.S.A). The glomerular filtration rate (GFR) value was determined with the Modification of Diet in Renal Disease criteria.Proteinuria was measured using the 24-h proteinuria levels. The patients were divided into four groups according to the amount of proteinuria in 24-h urine: proteinuria <200 mg/day, proteinuria 200-1000 mg/day, proteinuria 1000-3000 mg/day, and proteinuria >3000 mg/day (at nephrotic level).

Ambulatory BP measurements were conducted using noninvasive multitasking BP recorders (TM2425; A&D, Tokyo, Japan). Blood pressure was recorded at 15-min intervals between 07:00 and 21:00 and at 30-min intervals between 21:00 and 07:00. Mean systolic BP (SBP) and diastolic BP (DBP) values were calculated for each participant. Mean BP (MBP) was calculated as the sum of DBP and one-third of the pulse pressure. Daytime and nighttime BP were obtained as the mean values during daytime and nighttime, respectively. Daytime and nighttime BP ratios were then analyzed in each participant. Since the technique could cause errors, SBP >250 mmHg or <70 mmHg, DBP >130 mmHg or <30 mmHg, and pulse pressure >160 mmHg or <20 mmHg were not measured.

The patients were divided into three stages according to the BP levels specified in the 2018 European Society of Cardiology/European Society of Hypertension guidelines for HT.[7] Stage 1 HT was accepted as SBP 140-159 mmHg or DBP 90-99 mmHg, Stage 2 HT was accepted as SBP 160-179 or DBP 100-109, and Stage 3 HT was accepted as SBP ≥180 mmHg or DBP ≥110. In this classification made with the ABPM, a ≥10% decrease in the BP value measured at night compared to the daytime value was defined as dipper HT, a decrease of <10% was defined as non-dipper HT, and a nighttime increase in BP was defined as reverse-dipper HT.[8]

Statistical analysis

The data were evaluated with the IBM SPSS version 26.0 software (IBM Corp., Armonk, NY, USA). Results were expressed as mean ± standard deviation and median (min-max) for quantitative variables. Categorical data were presented as frequency (percentage). Normal distribution was examined with the Kolmogorov-Smirnov and Shapiro-Wilk tests. Three and above one-way variance test was used to compare normally distributed data according to groups. Multiple comparisons were examined with the Tamhane T2 and Duncan tests. The Mann-Whitney U test was used to compare nonnormally distributed data between two groups. The Kruskal-Wallis H test was used to compare nonnormally distributed data between three or more groups, and multiple comparisons were examined with the Dunn test with Bonferroni correction. Multiple comparisons were examined with the Bonferroni-corrected Z test. Nonnormally distributed data was examined with the Spearman rho correlation coefficient. Factors affecting the presence of proteinuria were examined by logistic regression analysis. A p-value <0.05 was accepted as statistically significant.

RESULTS

The median duration of HT was 60 (1-480) months. The mean creatinine level was 1.29±0.94 mg/dL, the mean GFR was 75.85±34.36 mL/min/1.732, and the mean 24-h urine protein was 829.8±1591.14 mg. Stage 1 HT was detected in 49.3% of the patients, Stage 2 HT was detected in 31%, and Stage 3 HT was detected in 19.7%. The mean 24-h SBP was 128 mmHg, daytime

Table 1
Demographic and laboratory characteristics of the patients (n=163)

	n	%	Mean±SD	Median	Min-Max
Age (year)			50.7±16.6		
Sex					
Female	79	48.5			
Male	84	51.5			
HT duration (month)				60	1-480
Stage 1 HT	35	49.3			
Stage 2 HT	22	31			
Stage 3 HT	14	19.7			
Creatinine (mg/dL)			1.29±0.94		
GFR (mL/min/1.73 m^2)			75.85±34.36		
Albumin			43.98±4.53		
24 h urine protein (mg/24 h)			829.8±1591.14		
Daytime DBP (mmHg)			80.26±11.94		
Nighttime DBP (mmHg)			76.78±13.23		
Daytime SBP (mmHg)			129.76±16.6		
24 h DBP (mmHg)			79.68±11.89		
Nighttime SBP (mmHg)			125.89±19.35		
24 h SBP (mmHg)			128.88±16.79		
24 h MBP (mmHg)			102.51±13.5		
Daytime MBP (mmHg)			103.26±13.32		
Nighttime MBP (mmHg)			99.36±14.76		
Dipper HT					
No	76	46.6			
Yes	87	53.4			
Non-dipper HT					
No	115	70.6			
Yes	48	29.4			
Reverse-dipper HT					
No	135	82.8			
Yes	28	17.2			

SD: Standard deviation; HT: Hypertension; GFR: Glomerular filtration rate; DBP: Diastolic blood pressure; SBP: Systolic blood pressure; MBP: Mean blood pressure.

Table 2
Comparison of laboratory and ABPM measurements of patients with and without proteinuria

	Group without proteinuria				Group with proteinuria				Statistic	p
	n	%	Mean±SD	Median (Min-Max)	n	%	Mean±SD	Median (Min-Max)		
Age (year)			46.96±17.15				53.2±16.75			
Sex										
Female	36	50			26	51				
Male	36	50			25	49				
Stage 1 HT	24a	68.6			7ab	35				
Stage 2 HT	8a	22.9			9a	45				
Stage 3 HT	3a	8.6			4ab	20				
Creatinine (mg/dL)				0.86 (0.4-1.83)b				0.98 (0.42-5)ab		
GFR (ml/min/1.73 m²)				95 (1.02-135)b				75 (8-149)ab		
Albumin				46 (30-52)c				44 (34-50)b		
Daytime DBP (mmHg)			78.2±10				79.98±12.89			
Night DBP (mmHg)			72.58±9.23b				77.52±14.44ab			
24 h DBP mmHg			76.99±9.31b				79.79±12.93ab			
Daytime SBP (mmHg)				124 (98-152)b				132 (92-182)ab		
Night SBP (mmHg)			117.35±10.67b				127.65±19.02a			
24 h SBP mmHg			122.77±11.18b				129.29±16.66ab			
24 h MBP (mmHg)			97.89±9.43b				103.45±13.93ab			
Daytime MBP (mmHg)			99.51±10.09b				103.49±13.51ab			
Night MBP (mmHg)				92 (67-121)b				102 (73-153)a		
Dipper HT	17	23.3			34	65.4				
No	56a	76.7			18b	34.6				
Yes										
Non-dipper HT	63	86.3			28	53.8				
No	10a	13.7			24b	46.2				
Yes										
Reverse-dipper HT	66	90.4			42	80.8				
No	7	9,6			10	19.2				
Yes										

	Group with proteinuria				Statistic	p
	n	%	Mean±SD	Median (Min-Max)		
Age (year)			53.71±15.18		2.106	0.069d
Sex					0.867	0.851f
Female	12	42.9				
Male	16	57.1				
Stage 1 HT	2b	18.2			14.634	0.014f
Stage 2 HT	4a	36.4				
Stage 3 HT	5b	45.5				
Creatinine (mg/dL)				1.44 (0.59-4.9)a	23.171	<0.001e
GFR (ml/min/1.73 m²)				55.5 (0.27-108)a	24.404	<0.001e
Albumin			44 (30-49)ab		30.223	<0.001e
Daytime DBP (mmHg)			84.36±14.59		2.378	0.072d
Night DBP (mmHg)			82.93±16.67a		8.008	0.031d
24 h DBP mmHg			84.39±14.82a		3.971	0.015d
Daytime SBP (mmHg)				131 (113-187)a	15.842	0.021e
Night SBP (mmHg)			138.86±25.36a		12.167	<0.001d
24 h SBP mmHg			138.86±20.53a		8.164	<0.001d
24 h MBP (mmHg)			109.36±17.04a		7.142	0.001d
Daytime MBP (mmHg)			109.5±16.89a		5.026	0.005d
Night MBP (mmHg)				108 (72-150)a	27.177	<0.001e
Dipper HT	18	64.3			29.610	<0.001f
No	10b	35.7				
Yes						
Non-dipper HT	18	64.3			17.489	0.001f
No	10ab	35.7				
Yes						
Reverse-dipper HT	20	71.4			7.235	0.057f
No	8	28.6				
Yes						

SD: Standard deviation; a-c: There is no difference between groups with the same letter (Bonferroni correction Z test, Dunn test, Tamhane's T2 test and Duncan test); d: One-way variance test; e: Kruskall Wallis H test, f: Fisher's exact test,

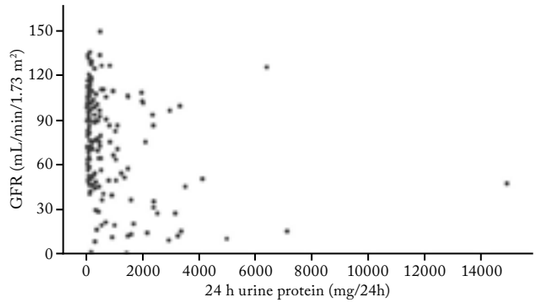

Figure 1. Relationship between proteinuria and GFR.
GFR: Glomerular filtration rate.

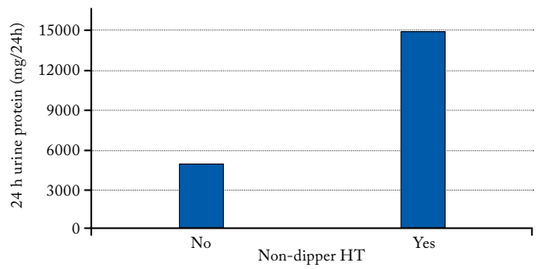

Figure 2. Relationship between proteinuria and non-dipper HT.
HT: Hypertension.

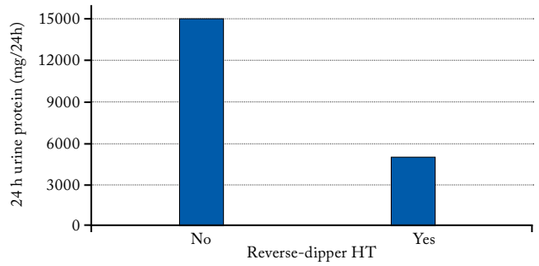

Figure 3. Relationship between proteinuria and reverse-dipper HT.
HT: Hypertension.

SBP was 129 mmHg, and nighttime SBP was 123 mmHg. The mean 24-h DBP was 79.68±11.89 mmHg, daytime DBP was 80.26±11.94 mmHg, and nighttime DBP was 76.78±13.23 mmHg. The mean 24-h MBP was 102.51±13.5 mmHg, daytime MBP was 103.26±13.32 mmHg, and nighttime MBP was 99.36±14.76 mmHg. Dipper HT was detected in 53.4% of the patients, non-dipper HT in 29.4%, and reverse-dipper HT in 17.2% (Table 1).

The dipper HT, albumin, and GFR were significantly lower in those with proteinuria compared to those without proteinuria. Age, creatinine, HT duration, 24-h SBP, daytime SBP, nighttime SBP, DBP, and MBP, non-dipper HT (all p<0.001), 24-h DBP (p=0.015), daytime MBP (p=0.005), and reverse-dipper HT (p=0.001) were significantly higher in the group with proteinuria (Table 2). A significant relationship was detected in the univariate regression analysis between proteinuria and creatinine, GFR (Figure 1), albumin, 24-h SBP, daytime SBP, nighttime SBP, daytime MBP, nighttime MBP, dipper HT, non-dipper HT (p<0.001 for all; Figure 2), reverse-dipper HT (p=0.025; Figure 3), and age (p=0.011). However, no relationship was detected with DBP. In multivariate logistic regression analysis, a relationship was found between proteinuria and albumin (p=0.027), night SBP (p=0.001), and 24-h SBP (p=0.028; Table 3). When the patients were divided into dipper, non-dipper, and reverse-dipper HT, the duration of HT was shorter and GFR and albumin were higher in those with dipper HT. Twenty-four-hour urine proteinuria, nighttime SBP, 24-h SBP, and nighttime MBP were lower in those with dipper HT.

DISCUSSION

In the present study, the SBP, DBP, and MBP values that were determined with ABPM were higher in hypertensive patients who had proteinuria. It was also found that the development of non-dipper HT and reverse-dipper HT was more frequent in those who had proteinuria. Creatinine and urine protein levels were higher and there were more advanced stages of HT in patients who had non-dipper HT and reverse-dipper HT.

Rational management of HT begins with accurate measurement of BP. European[7] and American[9] guidelines recommend the use of ABPM in all patients using antihypertensive medications. The reasons for this recommendation of these guidelines include the differential diagnosis of causes such as whitecoat HT, masked HT, orthostatic HT, chronic renal failure, autonomic dysfunction, diabetes mellitus, and endocrine HT, as well as determining the time of hypertensive drug use.

Table 3
Identifying factors associated with proteinuria using logistic regression analysis

	Univariate			Multivariate		
	OR	95% CI	*p*	OR	95% CI	*p*
Age	1.026	1.006-1.046	0.011	1.027	0.934-1.13	0.578
Sex						
Female	1.119	0.601-2.084	0.723			
HT duration (month)	1.006	1.001-1.010	0.015			
Creatinine (mg/dL)	4.311	1.992-9.327	<0.001	15.35	0.014-17398.799	0.447
GFR (mL/min/1.73 m²)	0.978	0.968-0.988	<0.001	1.037	0.93-1.156	0.511
Albumin	0.811	0.736-0.895	<0.001	0.756	0.59-0.968	0.027
Daytime DBP (mmHg)	1.028	0.999-1.057	0.055			
Night DBP (mmHg)	1.050	1.021-1.080	0.001			
Daytime SBP (mmHg)	1.041	1.017-1.065	0.001	2.771	0.573-13.409	0.205
Night SBP (mmHg)	1.059	1.033-1.084	<0.001	1.297	1.109-1.516	0.001
24 h SBP mmHg	1.048	1.024-1.074	<0.001	0.858	0.748-0.984	0.028
24 h DBP mmHg	1.038	1.008-1.068	0.012			
24 h MBP (mmHg)	1.055	1.026-1.086	<0.001			
Daytime MBP (mmHg)	1.044	1.016-1.073	0.002			
Night MBP (mmHg)	1.071	1.040-1.104	<0.001			
Dipper HT (Ref.: No)	0.160	0.080-0.320	<0.001	0.24	0.004-14.049	0.492
Non-dipper HT (Ref.: No)	4.604	2.095-10.118	<0.001	0.245	0.014-4.318	0.337
Reverse-dipper HT (Ref.: No)	2.870	1.144-7.197	0.025			

OR: Odds ratio; CI: Confidence interval: Wald backward stepwise method.

Additionally, a relationship was detected with the progression of microvascular diseases.[10] Ambulatory BP measurement was shown to be more effective in indicating the development of target organ damage.[11,12] Therefore, ABPM was used to determine the BP values of the patients in the present study. Furthermore, the ABPM was found to be the most useful and effective method in diagnosing HT, and it is also the best method in determining the time of taking antihypertensive medication.[7]

In the present study, the relationship between ABPM and 24-h urinary protein excretion was examined in patients who applied to the nephrology clinic with complaints of HT. Similar to the literature data, as the proteinuria level of the patients increased, an increase in creatinine levels and a decrease in GFR were detected.[13] The reason for this relationship was the increase in glomerular perfusion pressure, which may result in endothelial cell damage in the glomerular capillaries, resulting in microalbuminuria and progressive renal damage, similar to the study reported by Ying et al.[13]

Hermida et al.[14] reported that non-dipper or reverse-dipper HT was more common in patients who had resistant and uncontrolled HT. When the renal functions of dipper HT, non-dipper HT, and reverse-dipper HT patients were compared, GFR was found to be lower in patients who had non-dipper and reverse-dipper HT at significant levels. Hermida et al.[15] reported that non-dipper HT was more common in patients who had chronic renal failure, similar to our study, and GFR was lower and creatinine was elevated in those with non-dipper HT.

As a result of the increased BP at night, there is increased peripheral resistance and thickness of the glomerular basement membrane, which causes cell damage in the vascular endothelium, increasing albumin/protein excretion.[16] It was found in the present study that as proteinuria increased, dipper HT

and non-dipper HT developed in patients. Similar to our study, Farrag et al.[17] and Guo et al.[18] reported that the frequency of non-dipper HT increased as proteinuria increased and proteinuria was elevated in patients who had non-dipper HT. A recent study that investigated the bidirectional relationship of proteinuria and BP argued that proteinuria and BP might influence each other, suggesting that increase in proteinuria will cause higher BP and vice versa.[19] Similarly, the present study found that SBP, DBP, and MBP values that were determined with ABPM were elevated in patients who had proteinuria. Mettimano et al.[20] reported a significant relationship between proteinuria and 24-h SBP, daytime SBP, and nighttime SBP values. O'Seaghdha et al.[21] reported that this relationship was contradictory to DBP. Similar to our study, Hirayama et al.[22] reported a relationship between proteinuria and SBP but not with DBP. Differences between studies might be due to the differences in patient age. The ages of the patients in our study were higher compared to those in the Asian study and other referenced studies.[21,22] Low DBP levels reflect improved arterial stiffness in the elderly, which may be a risk factor associated with poor renal prognosis.[20] In other words, it may be a more practical method to follow up patients in the elderly with SBP.

This study had some limitations. First, since the study was conducted at a cross-sectional design, changes over time in the relationship between proteinuria and HT were not investigated. Second, proteinuria was assessed with 24-h urine collection, and spot urine protein-to-creatinine ratio was not examined. Third, the number of patients was insufficient since the study was conducted within a short period.

In conclusion, the development of non-dipper HT and reverse-dipper HT was more common in those with proteinuria compared to those without proteinuria. Renal dysfunction and proteinuria were more common in patients who have non-dipper or reverse-dipper HT. Advanced stage HT development was detected in those who have non-dipper HT and reverse-dipper HT compared to those with dipper HT. Ambulatory BP monitoring was more useful than other tests in hypertensive patients with proteinuria, and proteinuria could be controlled with strict BP control in such patients. However, further multicenter studies with a larger number of patients are needed.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea/concept, data collection and/or processing, literature review, writing the article, materials, other: B.A.D.; Design, analysis and/or interpretation, critical review, references and fundings: M.İ.D.; Control/supervision: A.Ö.

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