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Relationship Between Kidney Length and Cortical Thickness and Circadian Blood Pressure Measurements in Hypertensive Patients

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Introduction. It has been demonstrated that kidney length is associated with office blood pressure (BP) measurements. Several studies support that ambulatory BP measurements in comparison to conventional BP better correlate with hypertensive target organ damage, and that the lack of nocturnal dip in BP (nondipping) is related to an increase in the incidence of cardiovascular event in essential hypertensive patients. This study evaluated the specific relationship between kidney length, renal cortical thickness (RCT), and circadian BP in hypertensive patients.

Materials and Methods. In a cross-sectional study, 144 patients with newly diagnosed essential hypertension underwent physical examination, office BP measurements, laboratory analysis, ambulatory BP monitoring, renal ultrasonography, and spot and 24-hour urine collection.

Results. There were 103 dipper (71.5%) and 41(28.5%) nondipper patients. Among the dippers, 13 were extreme dippers and among nondippers 11 were reverse dippers. Most of the ambulatory BP measurements were not associated with kidney length or RCT. Kidney length and RCT were not different among dippers, extreme dippers, nondippers, and reverse dippers. The kidney length and RCT were not different between patients with white coat hypertension and sustained hypertension, either. Logistic regression analysis did not show any independent association between kidney length, RCT, and nondipping status.

Conclusions. Kidney length and RCT may not be associated with circadian BP monitoring.

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INTRODUCTION

The kidney plays a central role in electrolyte homeostasis and the regulation of blood pressure (BP). One mechanism by which kidneys might affect BP is through total filtration surface area and nephron mass, which are, in part, determined by glomerular number.[1,2] It has been speculated that nephron mass and glomerular number were related with kidney volume.[3] Kidney volume is related with kidney length.[4,5] In turn, several studies have shown that both kidney volume and kideny length were associated with BP.[1,6] It was proposed that congenital or programmed reduction in nephron number explains why some individuals are susceptible to hypertension and renal injury, whereas others are relatively resistant under similar circumstances. A reduction in nephron number and therefore whole-kidney glomerular surface area would result in reduced sodium excretory capacity, enhanced susceptibility to hypertension,

and reduced renal reserve, thereby limiting compensation for renal injury.[7] Indeed, it was found that patients with primary hypertension have lower nephron number compared to normotensives.[8]

Several studies support that ambulatory BP, in comparison to conventional BP, is better correlated with hypertensive target organ damage.[9] Additionally, ambulatory blood pressure monitoring (ABPM) along with office BP measurements allows defining hypertension subtypes such as white coat hypertension (WCHT), masked hypertension, and sustained hypertension (SHT). Thus, by the light of aforementioned data, the current study aimed to investigate the relationship between kidney length, renal cortical thickness (RCT), and ABPM and nondipping pattern in never-treated hypertensive patients.

MATERIALS AND METHODS

Participants

This cross-sectional study was in accordance with the declaration of Helsinki and approved by the local ethics committee. Informed consent was obtained from all patients before enrollment. The study population comprised of newly diagnosed patients with essential hypertension. Patients with secondary hypertension, liver disease, symptomatic heart failure, coronary heart disease, neurologic disorders or deficits, pulmonary diseases, autoimmune disease, endocrine disorders, malignancies, polycystic kidney disease, solitary kidneys, urinary tract infection, and menstruatation during the study period were not included. None of the patients had a history of acute coronary syndrome, peripheral revascularization, or amputation.

Basic Measurements

On 12-lead electrocardiography, all of the patients had normal sinus rhythm, no conduction disturbances, and no ST-T changes. None of the patients were shift workers or reported alcohol use. All of the patients underwent medical history taking, office BP measurement, calculation of body surface area (according to Du Bois and Du Bois formula),[10] physical examination, routine biochemistry measurements (after an overnight fasting), routine urinary analysis, 24-hour ABPM monitoring, and ultrasonography examination of the kidneys. The 24-hour urine specimens were collected to determine creatinine clearance as well as protein and albumin excretion. If the urine creatinine excretion of the two consecutive specimens differed by more than 10%, another 24-hour collection was made to assess the adequacy of collection.

Ambulatory Blood Pressure Measurement

Ambulatory 24-hour BP monitoring was performed on each patient's nondominant arm, using a Space Labs 90207 oscillometric monitor (Redmond, Washington, USA), concomitantly with ultrasonography (within 1 week). The accuracy of the device was checked against the standard auscultatory method to ensure that the difference in BP measurements between methods did not exceed +5 mm Hg. The device was set to obtain BP readings at 20-minute intervals during the day (07:00 AM to 11:00 PM) and at 30-minute intervals during the night (11:00 PM to 07:00 AM). Each ambulatory BP monitoring dataset was first automatically scanned to remove artifactual readings. Data were edited by omitting all readings of zero, all heart rate readings less than 20 or greater than 200 per minute, diastolic BP readings less than 40 mm Hg and greater than 150 mm Hg, systolic BP readings less than 70 mm Hg and greater than 240 mm Hg, and all readings where the differences between systolic and diastolic BPs was less than 10 mm Hg. Readings were evaluated if the percentage of successful readings were above 90%. All subjects were instructed to rest or sleep between 11:00 PM and 7:00 AM (nighttime) and to continue their usual activities between 7:00 AM and 11:00 PM (daytime). Patients were asked to remain still at the time of measurement and to note in a diary the occurrence of unusual events or poor sleep.

''Nocturnal dipping'' was defined as a reduction of greater than 10% (when compared with the daytime values) in the systolic or diastolic BP levels at night. Extreme nocturnal dipping was defined as reduction in average systolic BP and diastolic BP at night of 20% or greater when compared to daytime values. Reverse dipping was defined as higher nocturnal average systolic BP and diastolic PB in comparison with daytime values.[11] Patients were divided also into 2 separate groups as WCHT and SHT. The definition of WCHT and SHT were as follows respectively: office systolic and diastolic BP of 140/90 mm Hg and greater and daytime ambulatory BP less than 135/85 mm Hg and office systolic and diastolic BP of 140/90 and greater

and daytime ambulatory BP time BP of 135/85 mm Hg and greater.[12]

Renal Ultrasonography

The evaluation of bipolar kidney length and RCT was performed by a single radiologist who was blinded to any clinical information using a GE LOGIQ 5 PRO ultrasound system, with a 3.5-MHz curved array transducer (GE Healthcare, Waukesha, WI, USA). Kidney lengths were measured as the greatest pole-to-pole distance in the sagittal plane. The RCT was measured in the sagittal plane at the level of the mid-kidney as described by Moghazi and colleagues.[13] The measurement was taken over a medullary pyramid, perpendicular to the capsule as the shortest distance from the base of the medullary pyramid to the renal capsule. The reader was blinded to specific kidney function, additional imaging, or any additional clinical information at the time of image review. Cortical thickness and length were measured bilaterally.

Statistical Analysis

Statistical analysis was performed with the SPSS software (Statistical Package for the Social Sciences, version 15.0, SPSS Inc, Chicago, Ill, USA). Data was checked for normality. Data were shown as mean ± standard deviation and percentage (%) where appropriate. Comparisons of the two groups were assessed by means of the *t* test for normally distributed continuous variables and by the Mann-Whitney U test for non-normally distributed continuous variables. For the comparison of four groups (dippers, nondippers, extreme dippers, and reverse dippers), the Kruskal-Wallis test was used. For the analysis of categorical variables, the chi-square test was used. The Pearson correlation analysis was used for correlations. Finally, multivariable logistic regression analyses were performed to assess the independent effects of several variables on nocturnal dipping status. These effects were measured by odds ratios, and their 95% confidence intervals on the basis of logistic regression models. Results were considered statistically significant if the 2-tailed *P* value was less than .05.

RESULTS

Initially, 164 patients were enrolled. One patient with renal artery stenosis, 2 patients with elevated liver enzymes, 1 patient with adrenal mass, 3 patients with diabetes mellitus, 1 patients with polycystic kidney disease, 1 patient with solitary kidney, 2 patients with urinary tract infection, 2 patients menstruating during the study period, and 7 patients who were not willing to participate were excluded. The final study population was consisted of 144 patients. There were 103 dipper (71.5%) and 41 (28.5%) nondipper patients. Among dippers, 13 were extreme dippers and among nondippers 11 were reverse dippers. The comparative demographic, ambulatory BP, and laboratory parameters among dippers and nondippers are shown in Table 1.

The mean right kidney length, left kidney length, right RCT, and left RCT were 109.6 ± 8.5 mm, 111.5 ± 9.7 mm, 13.2 ± 2.1 mm, and 13.5 ± 2.1 mm, respectively. Right kidney length correlated with left kidney length (r = 0.632, $P < .001$), right RCT (r = 0.285, $P = .001$), left RCT (r = 0.295, $P < .001$), age (r = -0.224, $P = .007$), body surface area (r = 0.195, $P = .02$), average ambulatory systolic BP (night; r = -0.211, $P = .01$), serum high-density lipoprotein cholesterol (r = -0.241, $P = .004$), serum triglyceride (r = 0.201, $P = .02$), and creatinine clearance (r = 0.297, $P = .003$). Left kidney length correlated with RCT (r = 0.280, $P = .001$), left RCT (r = 0.428, $P < .001$), age (r = -0.173, $P = .04$), body surface area (r = 0.196, $P = .02$), serum creatinine (r = -0.165, $P = .048$), serum triglyceride (r = 0.164, $P = .049$), and creatinine clearance (r = 0.402, $P < .001$).

Right RCT correlated with left RCT (r = 0.562, $P < .001$), age (r = -0.335, $P < .001$), average ambulatory diastolic BP (day; r = 0.219, $P = .008$), total cholesterol (r = 0.183, $P = .03$), triglyceride (r = 0.239, $P = .005$), hemoglobin (r = 0.300, $P < .001$), and creatinine clearance (r = 0.333, $P = .001$). Left RCT correlated with age (r = -0.394, $P < .001$), average ambulatory diastolic BP (day; r = 0.171, $P = .04$), serum creatinine (-0.229, $P = .006$), blood urea nitrogen (-0.233, $P = .005$), serum potassium (r = -0.181, $P = .03$), serum total cholesterol (r = 0.235, $P = .005$), serum triglyceride (r = 0.169, $P = .04$), and creatinine clearance (r = -0.250, $P = .01$). Comparison of kidney length and RCT among dippers, extreme dippers, nondippers and reverse dippers are shown in Table 2.

Eigthy-seven patients (60.4%) had WCHT, whereas 57 patients (39.6%) had SHT. Comparison of right kidney length, left kidney length, right RCT, and left RCT between WCHT and SHT showed that right kidney length (110.0 ± 7.9 mm versus 108.4 ± 9.4

Table 1. Demographics, Ambulatory Blood Pressure (BP), and Laboratory Parameters Among Dippers and Nondippers

Parameter	Dippers (n = 103)	Nondippers (n = 41)	*P*
Age, y	55.5 ± 11.5	46.8 ± 11.3	< .001
Sex			
Male	37	19	
Female	66	22	.25
Body Mass Index	26.6 ± 3.2	27.1 ± 4.7	.56
Body Surface Area	1.77 ± 0.17	1.78 ± 0.15	.99
Smoker	41	15	.72
Clinical systolic BP, mm Hg	150.6 ± 12.1	157.1 ± 19.1	.02
Clinical diastolic BP, mm Hg	92.9 ± 8.7	96.9 ± 10.4	.03
Ambulatory systolic BP (day), mmHg	130.9 ± 13.2	137.5 ± 18.2	.02
Ambulatory diastolic BP (day), mm Hg	77.5 ± 11.2	79.4 ± 11.7	.36
Heart rate (day), beat/min	73.2 ± 11.6	75.5 ± 12.5	.29
Ambulatory systolic BP (night), mmHg	111.6 ± 11.6	131.4 ± 18.4	< .001
Ambulatory diastolic BP (night), mm Hg	64.3 ± 8.2	74.1 ± 12.2	< .001
Heart rate (night), beat/min	61.6 ± 10.5	66.8 ± 12.5	.01
Ambulatory systolic BP (24-h average), mmHg	126.8 ± 12.5	136.2 ± 17.9	< .001
Ambulatory diastolic BP (24-h average), mm Hg	74.2 ± 8.7	79.1 ± 11.1	.01
Heart rate (24-h average), beat/min	70.2 ± 10.8	73.7 ± 12.2	.10
Mean arterial BP (day), mm Hg	95.3 ± 10.8	99.6 ± 13.8	.03
Mean arterial BP (night), mm Hg	80.1 ± 7.5	94.1 ± 14.3	< .001
Mean arterial BP (24-h average), mm Hg	91.0 ± 9.1	98.3 ± 13.8	< .001
Fasting blood glucose, mmol/L	5.28 ± 0.76	5.30 ± 0.80	.87
Blood urea nitrogen, mmol/L	5.1 ± 1.6	6.4 ± 2.8	.002
Serum creatinine, μmol/L	76.9 ± 18.6	99.9 ± 47.7	.003
Serum albumin, g/L	46.2 ± 4.1	44.7 ± 3.8	.04
Hemoglobin, g/L	139.8 ± 11.7	136.9 ± 12.6	.19
Serum sodium, mmol/L	140.4 ± 3.1	140.5 ± 3.0	.82
Serum potassium, mmol/L	4.30 ± 0.33	4.35 ± 0.45	.45
Serum calcium, mmol/L	2.34 ± 0.12	2.33 ± 0.09	.39
Serum phosphorus, mmol/L	1.12 ± 0.17	1.13 ± 0.16	.78
Serum uric acid, μmol/L	296.2 ± 78.5	347.9 ± 96.4	.002
Alanine aminotransferase, μkat/L	0.35 ± 0.14	0.36 ± 0.14	.34
Aspartate aminotransferase, μkat/L	0.39 ± 0.16	0.35 ± 0.15	.69
Serum total cholesterol, mmol/L	5.65 ± 0.98	5.50 ± 0.82	.37
Low-density lipoprotein cholesterol, mmol/L	3.40 ± 0.81	3.25 ± 0.74	.36
High-density lipoprotein cholesterol, mmol/L	1.47 ± 0.33	1.49 ± 0.34	.57
Triglyceride, mmol/L	1.55 ± 0.74	1.54 ± 0.64	.86
Thyroid-stimulating hormone, mU/L	1.58 ± 1.03	1.44 ± 1.06	.36
High-sensitivity C-reactive protein, mg/L	0.98 ± 1.05	0.98 ± 0.66	.15
Creatinine clearance, mL/min/1.73m^2	91.8 ± 28.2	72.1 ± 30.2	.005
24-hour urine protein excretion, mg/d	134.7 ± 102.0	299.0 ± 385.1	.009
24-hour urine albumin excretion, mg/d	19.4 ± 63.4	91.4 ± 252.6	.04

Table 2. Comparison of renal length and renal cortex among dippers, extreme dippers, non-dippers and reverse dippers

Parameter	Dippers (n = 90)	Extreme Dippers (n = 13)	Nondippers (n = 30)	Reverse dippers (n = 11)	*P**
Right kidney length, mm	109.9 ± 8.3	110.9 ± 7.7	109.2 ± 8.7	103.7 ± 9.8	.31
Left kidney length, mm	111.6 ± 8.5	113.7 ± 9.2	111.4 ± 13.2	108.0 ± 9.4	.45
Right renal cortical thickness, mm	13.3 ± 2.1	13.4 ± 2.7	12.7 ± 2.0	12.7 ± 1.6	.32
Left renal cortical thickness, mm	13.6 ± 1.9	13.8 ± 2.1	13.1 ± 2.7	13.2 ± 2.0	.81

**P* values are based on the Kruskal-Wallis test.

mm, $P = .26$), left kidney length (111.6 ± 9.4 mm versus111.2 ± 10.3 mm, $P = .82$), right RCT (13.0 ± 1.9 mm versus 13.4 ± 2.2 mm, $P = .22$), and left RCT (13.3 ± 2.0 mm versus 13.8 ± 2.2 mm, $P = .18$) were not significantly different. Logistic regression analysis was performed to determine the association of the following independent factors with dipping and nondipping status (as a dependent variable): sex, age, smoking status, body mass index, plasma glucose, serum uric acid, serum total cholesterol, serum triglyceride, 24-hour urine proten excretion, 24-hour urine albumin excretion, 24-hour creatinine clearance, right kidney length, left kidney length, right RCT, left RCT. The results of logistic regression analysis are shown in Table 3.

DISCUSSION

The present study demonstrated that first, most of the ABPMs were not related with kidney length and cortical thickness; second, kidney length and cortical thickness were not different between dippers, nondippers, reverse dippers, and extreme dippers; third, kidney length and RCT were not different in patients with WCHT and SHT; and fourth, kidney length and cortical thickness were not independently associated with dipping or nondipping pattern.

Renal ultrasonography is a simple and useful technique to determine kidney length, RCT, and echogenicity of kidneys.[14] It was also suggested that the bipolar kidney length is the most practical measurement of kidney size.[15] The prognostic importance of kidney size can be explored by evaluating the cross-sectional and longitudinal relationship of kidney size with nephron mass in health and in disease. Cross-sectional studies demonstrate that kidney size is related to renal parenchymal mass, renal volume, nephron number, and size of the nephrons.[4,5]

Various studies have shown that nephron number (which is related with renal mass and kidney length)[3] is associated with BP. Renal length and renal cortical thickness have also been associated with BP.[1,6] It was shown that patients with primary hypertension have lower nephron number compared to normotensives.[8] Similarly, among Australian Aboriginals, glomeruli numbers were lower among those with hypertension.[16] Brenner and colleagues proposed that congenital or programmed reduction in nephron number results in reduction of whole-kidney glomerular surface area, resulting in reduced sodium excretory capacity, leading to hypertension.[7] In support of the low nephron number, hypothesis, nephron deficient rats develop spontaneous hypertension, which is salt sensitive, exhibit greater levels of albuminuria, lower glomerular filtration rate and sodium excretion, and higher tissue sodium content, as compared with normal controls.[17,18]

Since ambulatory BP measurement is superior to office BP measurements, one can suggest that the relationships between kidney length and cortical thickness (which were hypothesized to be related with renal mass and glomerular number) with BP will even become stronger by using ABPM. Thus, the current study tested the hypothesis that kidney length and RCT measured by ultrasonography

Table 3. Logistic Regression for Factors Associating With Nocturnal Nondipping

Factor	Odds Ratio	95% Confidence Interval	*P*
Male sex	0.417	0.096 to 1.812	.24
Age	1.119	1.038 to 1.209	.003
Smoking Status	2.170	0.557 to 8.461	.26
Body mass index	1.192	0.980 to 1.449	.08
Fasting blood glucose	1.009	0.952 to 1.068	.77
Serum uric acid	1.739	1.039 to 2.906	.04
Total cholesterol	1.010	0.991 to 1.029	.31
Triglyceride	0.994	0.984 to 1.005	.31
24-hour urine protein excretion	1.013	1.003 to 1.024	.02
24-hour urine albumin excretion	1.018	1.002 to 1.035	.03
24-hour creatinine clearance	1.009	0.982 to 1.037	.50
Right renal length	0.944	0.862 to 1.034	.22
Left renal length	1.075	0.984 to 1.174	.11
Right renal cortical thickness	0.909	0.604 to 1.368	.65
Left renal cortical thickness	0.801	0.545 to 1.177	.26

is closely related with ABPM measurements. Surprisingly, however, the study did not show independent relationship between kidney length, renal cortical thickness, dipping or nondipping, or with almost none of the ABPM. There may be several explanations of not finding these relationships. Although traditionally it was accepted that kidney length correlates well with kidney function,[19] some authors suggest that not the kidney length, but renal volume is more sensitive in detecting kidney abnormalities.[20] It was also suggested that kidney length decreases with age as the kidney becomes thicker and wider, whereas kidney volume is stable with relatively little change.[21] Additionally, a small kidney, may be a surrogate for low nephron number, but growth in kidney size on ultrasonography cannot distinguish between normal growth with age and hypertrophy, potentially confounding this association.[22] Thus, the lack of association between kidney length and ABPM may be explained in the context of bipolar kidney length may not be a surrogate marker of renal mass and nephron number.

Furthermore, it was stated that not the glomerular numbers, but individual glomerular volume is more important with respect to kidney function and hypertension. It was speculated that low nephron number alone, does not account for all experimentally programmed hypertension.[23,24] In preterm infants it was found that the lower the glomeruli number, the higher the glomerul size.[25] Another study confirmed a significant direct correlation between nephron number and birth weight and a strong inverse correlation between glomerular volume and nephron number in both black and white neonates.[26] Another study showed a significant positive correlation between birth weight and nephron number with consistent inverse correlation between nephron number and glomerular size.[27] It was suggested that glomerular volume consistently varies inversely with nephron number, suggesting that larger glomeruli reflect compensatory hyperfiltration and hypertrophy that occur when nephron number is reduced. Glomerular size may therefore be an independent or additional risk factor predisposing to hypertension and kidney disease potentially affected by other programmable factors such as modulation of glomerular flow and salt sensitivity.[22] Lastly, various others factors such as altered expression of renal sodium transporters or modulation of the renin-angiotensin-aldosterone system, glomerulomegaly, gene polymorphisms, and maternal gestational hyperglycemia may all affect nephron number and susceptibility to hypertension.[22,28]

The current study has limitations that deserve to mention. Firstly, since the study has a cross-sectional design, cause and effect relationships cannot be suggested. Secondly, the study population is relatively small. However, study population composed of patients who were hitherto treated and have no other medical conditions potentially ruling out the effect of medication and other comorbidities. Thirdly, since antihypertensive medication may affect ambulatory BP recordings and the dipping phenomenon, extrapolation of these results to other populations (eg, patients with essential hypertension that use antihypertensive medication) is limited. Fourthly, the measurements were made for only once and serial measurements would give more information. Fifth, there is no control group. Lastly, kidney volume was not measured specifically in the current study.

CONCLUSIONS

The kidney length and cortical thickness did not associate with circadian BP monitoring. Other parameters such as glomerular volume, altered expression of renal sodium transporters or modulation of the renin-angiotensin-aldosterone system, glomerulomegaly, gene polymorphisms, maternal gestational hyperglycemia may all affect the relationship between renal volume and circadian BP. It should also be determined that whether other parameters related with hypertension such as carotid intima media thickness (as a measure of atherosclerotic process were associated with kidney length and RCT.[29-31]

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CONFLICT OF INTEREST

None declared.

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