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Changes in Ambulatory Blood Pressure Monitoring Parameters in Patients with Hypertension and Chronic Kidney Disease

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Abstract

Background: Current research has indicated that 24-hour ambulatory blood pressure monitoring (ABPM) is more effective than clinic blood pressure (BP) assessment in predicting cardiovascular outcomes and targeting organ damage. Our study aimed to analyze 24-hour ABPM patterns in patients with arterial hypertension (AH) and chronic kidney disease (CKD).

Methods and Results: This retrospective study included 1,000 patients (440 men and 560 women) aged 62.81±10.31 years with AH grades 1-3 (ESC/ESH, 2018) and CKD stages G1-G4. CKD stages were classified based on the GFR category (G1, G2, G3a, G3b, G4) (KDIGO 2012). All patients underwent an assessment of traditional risk factors, physical examination, clinical and biochemical laboratory methods, 12-lead ECG, and echocardiography. The 24-hour ABPM was performed using a Medicom Combi device (Russia). Depending on renal function, AH patients were divided into three groups: Group 1 included 220 AH patients with CKD G1 (eGFR>90 mL/min/1.73 m^2), Group 2 group included 512 AH patients with CKD G2 (eGFR 60-89 mL/min/1.73 m^2), and Group 3 included 268 AH patients with CKD G3a-G4 (eGFR 15-59 mL/min/1.73 m^2).

The average 24-hour SBP and DBP, daytime SBP, and nighttime SBP and DBP increased with worsening CKD stage with maximal values in CKD G3a-G4 (P=0.0000 in all cases). Nocturnal SBP decline was reduced to a greater extent in CKD G3a-G4 (P=0.0000). When assessing nocturnal SBP decrease (dipping), we found a "non-dipper" variant prevailed in all CKD groups. Patients with CKD G3a-G4 had a higher percentage of non-dipping status than those with CKD G1 (58.3% vs. 56.9%), and a “riser” variant was also higher (25.2% vs. 22.0%). Still, the relationship between the CKD stage and dipping status was not statistically significant (P>0.05 in both cases).

Conclusion: The worsening CKD stage in AH patients is associated with a severely impaired diurnal blood pressure profile. The presence of CKD requires the mandatory use of ABPM for correct diagnosis and assessment of cardiovascular disease risk. **(International Journal of Biomedicine. 2024;14(3):401-405.)**

Keywords: arterial hypertension • chronic kidney disease • ambulatory blood pressure monitoring • diurnal blood pressure profile

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Abbreviations

AH, arterial hypertension; **ABPM,** ambulatory blood pressure monitoring; **BP**, blood pressure; **BPV**, BP variability; **BMI**, body mass index; **CAD**, coronary artery disease; **CVD**, cardiovascular disease; **CKD**, chronic kidney disease; **DBP**, diastolic BP; **DBPV**, DBP variability; **DBPP**, diurnal blood pressure profile; **eGFR**, estimated glomerular filtration rate; **FBG**, fasting blood glucose; **HDL-C**, high-density lipoprotein cholesterol; **LDL-C**, low-density lipoprotein cholesterol; **PP**, pulse pressure; **SBP**, systolic BP; **SBPV**, SBP variability; **TC**, total cholesterol; **TG**, triglycerides; **T2D**, type 2 diabetes.

Introduction

Arterial hypertension (AH) is a leading risk factor for cardiovascular disease (CVD). The prevalence of hypertension in the adult population is 30–45%.[1] According to the national registry STEPS19, patients with hypertension account for approximately 38% of the population in Uzbekistan.[2] High blood pressure is a leading risk factor for the development of several CVD and chronic kidney disease (CKD).[3] Hypertension and diabetes are the primary causes of kidney damage. According to

social registers, the prevalence of kidney dysfunction without CVD and diabetes is 6.8%, with hypertension - 15.2%, and in combination with hypertension and diabetes - 43%.[4]

In AH patients with CKD, there is an increased risk for cardiovascular disease, progression of CKD, and all-cause mortality. The 2017 ACC/AHA High Blood Pressure Guideline recommends new thresholds and targets for diagnosing and treating hypertension in patients with and without CKD. A new aspect of the guidelines is the recommendation for measurement of out-of-office BP to confirm the diagnosis of hypertension and guide therapy.[5] In AH patients with CKD, ambulatory BP is a stronger predictor of targeting organ damage, kidney failure, cardiovascular events, and all-cause mortality than office BP pressure assessment.[6-9]

The purpose of the present study was to analyze 24-hour ambulatory blood pressure monitoring (ABPM) patterns in AH patients with CKD.

Materials and Methods

This retrospective study was performed in the Republican Specialized Scientifically Practical Medical Center of Cardiology and included 1000 patients (440 men and 560 women) aged 62.81±10.31 years with AH grades 1-3 (ESC/ESH, 2018)[10] and CKD stages G1-G4. CKD stages were classified based on the GFR category (G1, G2, G3a, G3b, G4) (KDIGO 2012).[11]

The exclusion criteria were symptomatic hypertension, cerebrovascular accidents, chronic heart failure (NYHA FC>III), and cardiac arrhythmia.

All patients underwent the following examinations: assessment of traditional risk factors, physical examination, clinical and biochemical laboratory methods, 12-lead ECG, echocardiography, and 24-hour ABPM.

Office BP was measured using a mercury sphygmomanometer, according to Korotkov's method. The 24-hour ABPM was performed in 415 patients using a Medicom Combi device (Russia). BP was measured during the daytime (07:00–23:00) every 30 min and at night (23:00–07:00) every 60 min. The interpretation of the results was based on generally accepted recommendations for ABPM quality criteria: monitoring duration of at least 23 hours, 50 successful measurements, and no "gaps" in the record lasting more than 1 hour.

Blood levels of lipids, urea, uric acid, glucose, HbA1C, and creatinine were determined using a Daytona autoanalyzer (RANDOX, UK). The estimated glomerular filtration rate (eGFR) was calculated according to the CKD-EPI (2021) equation.

Nine hundred sixty (96%) patients had chronic coronary artery disease (CAD), and 370(37%) had type 2 diabetes (T2D). A total of 572(57.2%) patients were obese, whereas 329(32.9%) were overweight. The average BMI of the patients was 31.50±5.73 kg/m^2. The clinical characteristics of the study population are summarized in Table 1.

All patients received combined cardioprotective and antihypertensive therapy, and patients with T2D received glucose-lowering treatment. The average office SBP/DBP on antihypertensive therapy was 133.94±20.01/83.64±10.36 mmHg. The laboratory and instrumental data of the study patients are shown in Table 2.

Table 1.

Clinical characteristics of AH Patients with CKD.

Variable	M±SD, n (%)
Age, years	62.81±10.31
Male, n (%)	440 (44%)
Female, n (%)	560 (56%)
Coronary artery disease, n (%)	960 (96%)
Diabetes mellitus, n (%)	370 (37%)
Obesity, n (%)	572 (57.2%)
Overweight, n (%)	329 (32.9%)
BMI, kg/м^2	31.50±5.73

Table 2.

Laboratory and instrumental characteristics of AH patients with CKD.

Variable	General group (n=1000) M±SD Me [Q1; Q3]
Office SBP, mmHg	133.94±20.01
Office DBP, mmHg	83.64±10.36
Creatinine, μmol/L	85.00 (70.00-100.00)
eGFR, ml/min/1.73 m^2	78.00 (57.00-86.00)
Urine albumin, mg/L	26.50 (12.60-46.80)
TC, mg/L	184.00 (154.00-216.00)
TG, mg/L)	153.00 (106.00-234.00)
LDL-C, mg/dL	104.00 (74.00-130.75)
HDL-C, mg/dL	40.00 (34.00-47.75)
FBG, mmol/L	5.30 (4.90-6.50)

Statistical analysis was performed using the statistical software package SPSS version 24.0 (SPSS Inc, Armonk, NY: IBM Corp). Baseline characteristics were summarized as frequencies and percentages for categorical variables and as median (Me) and interquartile range (IQR [Q1; Q3]), mean ± standard deviation (SD) for continuous variables. Multiple comparisons were performed using a one-way ANOVA and Tukey HSD post-hoc test. Group comparisons with respect to categorical variables were performed using the chi-square test. A probability value of $P<0.05$ was considered statistically significant.

Results

Depending on renal function, AH patients were divided into three groups: Group 1 included 220 AH patients with CKD G1 (eGFR>90 mL/min/1.73 m^2), Group 2 group included 512 AH patients with CKD G2 (eGFR 60-89 mL/min/1.73m^2), and Group 3 included 268 AH patients with CKD G3a-G4 (eGFR 15-59 mL/min/1.73 m^2).

The stage of CKD climbed steadily as age increased: 55.12±1.50, 63.21±1.68, and 68.91±3.94 years for CKD G1, CKD G2, and CKD G3a-G4, respectively (*P*=0.0000). As CKD stage increased (CKD G1, CKD G2, CKD G3a-G4), there was a steadily increase in BMI (30.65±0.68, 32.35±1.17, and 33.31±2.1 kg/m^2, respectively, *P*=0.0000), SBP (129.73±2.06, 130.29±1.9, and 144.00±11.37 mmHg, respectively, *P*=0.0000), DBP (84.59±1.13, 84.57±1.18, 88.57±4.59 mmHg, respectively, *P*=0.0000), as well as in the frequency of CAD (86.8%,97.1%, and 98.9%, respectively, *P*=0.0000) and T2D (28.2%, 36.9%, and 47.0%, respectively, *P*<0.0001) (Table 3).

Table 3.

Clinical, laboratory, and instrumental characteristics of AH patients depending on the CKD group.

	CKD G1 (n=220) [1]	CKD G2 (n=512) [2]	CKD G3a-4 (n=268) [3]	Statistics
Age, years	55.12±1.50	63.21±1.68	68.91±3.94	F=40242.84 P=0.0000 P_{1-2}=0.0000 P_{1-3}=0.0000 P_{2-3}=0.0000
BMI, kg/m^2	30.65±0.68	32.35±1.17	33.31±2.1	F=219.02 P=0.0000 P_{1-2}=0.0000 P_{1-3}=0.0000 P_{2-3}=0.0000
CAD, n (%)	191 (86.8)	497 (97.1)	265 (98.9)	χ2=46.589 df=2 P=0.0000
T2D, n (%)	62 (28.2)	189 (36.9)	126 (47.0)	χ2=18.521 df=2 P<0.0001
SBP, mmHg	129.73±2.06	130.29±1.9	144.00±11.37	F= 505.75 P=0.0000 P_{1-2}=0.4922 P_{1-3}=0.0000 P_{2-3}=0.0000
DBP, mmHg	84.59±1.13	84.57±1.18	88.57±4.59	F= 235.78 P=0.0000 P_{1-2}=0.9948 P_{1-3}=0.0000 P_{2-3}=0.0000
Creatinine, µmol/L	60.37±21.9	81.27±46.1	124.61±57.7	F=131.88 P=0.0000 P_{1-2}=0.0000 P_{1-3}=0.0000 P_{2-3}=0.0000
eGFR, mL/min/1.73 м^2	91.14±3.81	72.49±2.67	46.58±2.56	F=14444.10 P=0.0000 P_{1-2}=0.0000 P_{1-3}=0.0000 P_{2-3}=0.0000
Urine albumin, mg/L	40.74±8.8	40.03±14.83	72.35±68.30	F=73.35 P=0.0000 P_{1-2}=0.9694 P_{1-3}=0.0000 P_{2-3}=0.0000

An analysis of ABPM parameters showed that the average 24-hour SBP and DBP, daytime SBP, and nighttime SBP and DBP increased with increasing CKD stage and had maximal values in CKD G3a-G4 (*P*=0.0000 in all cases).

Nocturnal SBP decline was reduced to a greater extent in CKD G3a-G4 (*P*=0.0000). When assessing nocturnal SBP decrease (dipping), we found a "non-dipper" variant prevailed in all CKD groups. Patients with CKD G3a-G4 had a higher percentage of non-dipping status than those with CKD G1 (58.3% vs. 56.9%), and a "riser" variant was also higher (25.2% vs. 22.0). Still, the relationship between the CKD stage and dipping status was not statistically significant (*P*>0.05 in both cases) (Table 4). The levels of SBPV and DBPV were within the established threshold levels[12] daytime SBPV – 15 mmHg, daytime DBPV – 14 mmHg, and nighttime SBPV and DBPV – 15 mmHg and 12 mmHg, respectively. However, the values of daytime DBPV and nighttime SBPV increased with the worsening CKD stage.

Discussion

The results of many studies confirm that high BP is an important mechanism that leads to CKD. CKD remains one of the most important problems in public healthcare, as it is widespread and accompanied by various complications. Data on the CKD distribution in the Uzbek population are limited. We estimated the rate of CKD registration in the group of patients with hypertension at high and very high risks. Hypertension in patients with CKD shows the distinguishing features of ABPM. In our study, the worsening CKD stage was associated with a steady increase in clinic SBP and DBP. An analysis of ABPM parameters showed that the average 24-hour SBP and DBP, daytime SBP and DBP, and nighttime SBP and DBP increased with increasing CKD stage and had maximal values in CKD G3a-G4. The worsening CKD stage was associated with an impaired diurnal blood pressure profile. A "non-dipper" variant prevailed in all AH patients with CKD. Non-dipping is frequent in CKD and has also been consistently associated with increased CVD risk. Patients with CKD G3a-G4 had a higher percentage of non-dipping status than those with CKD G1 (58.3% vs. 56.9%), and a "riser" variant was also higher (25.2% vs. 22.0%) without statistically significant differences (*P*>0.05 in both cases). Most importantly, the prevalence of the riser BP pattern is associated with the highest CVD risk among all possible BP patterns. In addition, our patients also presented significantly elevated ambulatory PP, reflecting increased arterial stiffness and enhanced CVD risk.

In the AASK Cohort Study, among 617 patients with hypertension and GFR between 20 and 65 mL/min per 1.73 m^2, 498 participants (80%) had a non-dipping or reverse dipping profile. Of the 377 participants with controlled clinic BP (61%), 70% had masked hypertension.[13] A cross-sectional study performed by Mojón et al.[14] involved 10271 hypertensive patients with a mean age of 58.0±14.2 years. Among the participants, 3227 had CKD. The prevalence of non-dipping was significantly higher in patients with than without CKD (60.6% vs. 43.2%; *P*<0.001). The largest difference between groups was in the prevalence of the riser BP pattern (17.6% vs. 7.1% in patients with and without CKD, respectively; *P*<0.001). The riser BP pattern significantly and progressively increased from 8.1% among those with CKD G1 to 34.9% of those with CKD G5.[7] These results were consistent with our findings.

Table 4.

The 24-hour ABPM indicators depending on the CKD group.

Variable	CKD G1 (n=109) [1]	CKD G2 (n=203) [2]	CKD G3a-G4 (n=103) [3]	Statistics	
Average 24-h SBP, mmHg	120.09±11.14	124.58±12.11	146.29±7.57	F=4182.9027 P=0.0000 P_{1-2}=0.0016 P_{1-3}=0.0000 P_{2-3}=0.0000	
Average 24-h DBP, mmHg	71.16±8.41	72.56±8.24	74.86±11.11	F=4993 P=0.0117 P_{1-2}=0.3996 P_{1-3}=0.0089 P_{2-3}=0.0921	
Average 24-h PP, mmHg	48.92±8.71	52.02±10.89	71.43±11.18	F=152.3357 P=0.0000 P_{1-2}=0.0340 P_{1-3}=0.0000 P_{2-3}=0.0000	
Average daytime SBP, mmHg	121.98±11.11	124.94±11.95	146.43±7.11	F=173.6826 P=0.0000 P_{1-2}=0.0533 P_{1-3}=0.0000 P_{2-3}=0.0000	
Average daytime DBP, mmHg	72.98±8.56	73.78±8.90	76.29±11.84	F=1.5815 P=0.2069 P_{1-2}=0.7639 P_{1-3}=0.1896 P_{2-3}=0.3982	
Average nighttime SBP, mmHg	114.62±13.14	121.02±15.42	145.57±10.41	F=154.9383 P=0.0000 P_{1-2}=0.0003 P_{1-3}=0.0000 P_{2-3}=0.0000	
Average nighttime DBP, mmHg.	66.86±9.21	69.30±8.59	70.86±10.70	F=5.0306 P=0.0069 P_{1-2}=0.0713 P_{1-3}=0.0054 P_{2-3}=0.3500	
Nocturnal SBP decline, %	4.72±0.63	6.46±0.81	3.87±0.67	F=483.3190 P=0.0000 P_{1-2}=0.0000 P_{1-3}=0.0000 P_{2-3}=0.0000	
«Dipper», n (%)	21 (19.2)	52 (25.6)	16 (15.5)	χ^2=4.54 df=2 P=0.1033	χ^2=8.84 df=6 P=0.1828
«Non-dipper», n (%)	62 (56.9)	116 (57.1)	60 (58.3)	χ^2=0.048 df=2 P=0.9763	
«Riser», n (%)	24 (22.0)	30 (14.8)	26 (25.2)	χ^2=5.552 df=2 P=0.0632	
«Extreme-dipper», n (%)	2 (1.8)	5 (2.5)	1 (1.0)	χ^2=0.812 df=2 P=0.6663	
Average daytime SBPV, mmHg	12.95±4.06	14.06±4.97	14.38±7.36	F=2.1140 P=0.1221 P_{1-2}=0.2022 P_{1-3}=0.1385 P_{2-3}=0.8788	
Average daytime DBPV, mmHg	10.57±4.89	11.82±5.13	12.29±5.06	F=3.4197 P=0.0337 P_{1-2}=0.0943 P_{1-3}=0.0361 P_{2-3}=0.7221	
Average nighttime SBPV, mmHg	11.95±4.75	11.00±3.56	12.22±4.24	F=3.7723 P=0.0238 P_{1-2}=0.1222 P_{1-3}=0.8796 P_{2-3}=0.0363	
Average nighttime DBPV, mmHg	9.86±4.1	9.94±3.91	9.86±2.34	F=0.0250 P=0.9753 P_{1-2}=0.9811 P_{1-3}=0.9948 P_{2-3}=0.9818	

Conclusion

ABPM shows a pronounced impaired diurnal blood pressure profile in AH patients with CKD. The worsening CKD stage in AH patients is associated with a severely impaired diurnal blood pressure profile. The presence of CKD requires the mandatory use of ABPM for correct diagnosis and assessment of CVD risk.

Competing Interests

The authors declare that they have no competing interests.

Ethical Considerations

The study protocol was reviewed and approved by the Ethics Committee of the Republican Specialized Centre of Cardiology. The data was only used for study purposes without individual details identifying the patient.

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