

Nighttime Blood Pressure Abnormalities in Iranian CKD Patients: Necessity to Perform Ambulatory Blood Pressure Monitoring

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Introduction. Ambulatory blood pressure monitoring (ABPM) is a valuable tool for detecting abnormalities in nighttime blood pressure (BP), including non-dipping and nighttime hypertension. These abnormalities are independent predictors of a poor prognosis in patients with chronic kidney disease (CKD). The aim of our study was to analyze ABPM data and evaluate nighttime BP abnormalities in an Iranian CKD population.

Methods. This cross-sectional study was conducted on sixty-two patients at stages III and IV of CKD who were referred to a nephrology clinic in Tehran, Iran. The patients were classified as either dippers (19.4%) or non-dippers (80.6%), as well as nighttime normotensives (38.7%) or hypertensives (61.3%), based on ABPM data and in accordance with 2023 ESC/ESH guidelines. We compared demographic data, estimated glomerular filtration rate (eGFR), and daytime BP levels among these groups.

Results. The mean age of patients was 56.34 years, with 61.1% of them being male. Daytime pulse pressure was significantly greater in non-dippers compared to dippers (52.67 vs. 44 mmHg, P = .02). We found a significant correlation between the extent of BP dipping and eGFR (R = 0.281, P = .02). Systolic and diastolic daytime BP levels were significantly higher in individuals with nighttime hypertension. Diabetic patients were more likely to be non-dippers and have nighttime hypertension. After adjusting for age, diabetes mellitus, and daytime pulse pressure in a multivariable model, we determined that eGFR independently predicted the extent of BP dipping.

Conclusion. Our results showed that both non-dipping and nighttime hypertension are highly prevalent in CKD patients, but they have distinct contributing factors. The eGFR was identified as an independent predictor of BP dipping, whereas nighttime BP levels were primarily determined by daytime BP levels.

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INTRODUCTION

Elevated blood pressure (BP) and chronic kidney disease (CKD) are closely related disorders. Hypertension plays a significant role in the

development and progression of CKD. On the other hand, as CKD progresses, various factors contribute to the development of hypertension¹. Consequently, the prevalence of hypertension increases from

35% in stage I to over 80% in stages IV and V of CKD.² Additionally, high BP is associated with increased mortality among CKD patients³. The significance of managing BP in CKD patients is underscored by the fact that for every 10 mmHg increase in systolic BP, the risk of adverse events rises by approximately 16%.⁴ Therefore, it is crucial to control BP in CKD patients⁵. In line with this, the recent guideline by Kidney Disease: Improving Global Outcomes (KDIGO) recommends lowering the target systolic BP to less than 120 mmHg for CKD patients who do not undergo hemodialysis.⁶

In general practice, BP is typically assessed by using office measurements. However, these measurements only detect BP levels over a short period of time. In contrast, ambulatory blood pressure monitoring (ABPM) measures BP multiple times, allowing for the calculation of average BP levels throughout the day and night. Additionally, ABPM offers valuable information about the circadian rhythm of BP. Normally, BP follows a circadian rhythm, with a decrease of approximately 10 to 20% during nighttime, known as normal dipping.⁷

Studies have revealed a stronger correlation between ambulatory BP levels and adverse outcomes in patients with CKD compared to office BP measurements.⁸⁻¹⁰ ABPM can help to identify individuals with "masked" and "white coat hypertension", who face different risks for cardiovascular events and mortality.¹¹ Furthermore, ABPM provides valuable data on abnormal patterns of BP dipping, which independently predict poor prognosis and progression of kidney failure in CKD patients.^{10,12} Additionally, 24-hour monitoring of BP reveals patients with nighttime hypertension. Research suggests that nocturnal BP may be a better indicator of a worse prognosis than daytime BP levels.¹³

Most studies evaluating ABPM data in the Iranian population have focused on comparing ABPM results with office or home BP measurements. 14-17 Despite the higher prevalence of CKD in Iran compared to the global, there have been limited studies on ABPM data in these patients. 18 Rezvani *et al.* assessed the relationship between the circadian rhythm of BP and the severity of kidney failure. Since the mean eGFR of their study population was 66.03 cc/min/m², it appears that most patients were in the earlier stages of CKD. 19

Furthermore, no study has evaluated nighttime hypertension and its related factors in the general population, specifically in CKD patients, in Iran.

In this study, we aimed to assess ambulatory BP measurements and evaluate nighttime BP abnormalities, including non-dipping patterns and nocturnal hypertension, in a population with CKD in Iran. We also aimed to identify the factors contributing to these abnormalities.

MATERIALS AND METHODS Ethical Issues

The study protocol was designed in accordance with the Declaration of Helsinki and approved by the ethics committee of Tehran University of Medical Sciences (approval code IR.TUMS.IKHC. REC.1401.104). Patients were required to sign a written informed consent form in order to undergo ABPM and participate in the study.

Study Design

The research question was: What is the prevalence of abnormal patterns of nighttime BP in CKD patients, and how are these patterns influenced by other factors? Consequently, an analytical cross-sectional study was designed to investigate two important abnormalities in nighttime BP, including nighttime hypertension and impaired BP dipping, in a CKD population in Iran. The study also assessed the association of these abnormalities with parameters such as demographic data, stage of CKD, underlying diabetes mellitus, and daytime BP levels. Convenient sampling was used for selecting participants.

Participants

The study population consisted of CKD patients referred for routine follow-up to the Nephrology Clinic at Imam Khomeini Hospital Complex in Tehran, Iran, from July to December 2021. Patients who accepted the invitation to participate in the study and signed the informed consent were selected based on inclusion and exclusion criteria.

CKD was diagnosed on the basis of a serum creatinine level that remained elevated for more than three months,²⁰ or if the increase in serum creatinine was accompanied by small kidney size as reported in ultrasonography evaluation.^{20,21} The estimated glomerular filtration rate (eGFR) was calculated by using the CKD-EPI formula, and

considering the serum creatinine level, age, and sex of the patients.²² The inclusion criteria for the study were patients aged eighteen or older with stage III or IV chronic kidney disease (CKD), who had provided written informed consent. Those who were on renal replacement therapy and patients with congestive heart failure or decompensated cirrhosis were excluded.

Sample Size Calculation

The sample size calculation was based on the primary variable of abnormal BP dipping. Assuming $\alpha = 0.95$ and $\beta = 0.8$, approximately 57 cases were needed to detect a 70% prevalence in abnormal BP dipping with a precision error of 12%.²³ During the study period, 62 cases were enrolled.

Ambulatory BP Monitoring

Ambulatory 24-hour BP measurement was conducted by using the oscillometric method with the Microlife WatchBP O3 device (Microlife AG, Windau, Switzerland). This fully automated device has been validated by the European Society of Hypertension (ESH) and the Association for the Advancement of Medical Instrumentation (AAMI) protocols. 24,25 An appropriately sized cuff was applied to the non-dominant hand of each patient. The automated device was programmed to measure BP every 30 minutes during the day and every one hour during the night. The daytime and nighttime records were determined according to the patients' reported daily schedule. The ABPM was considered adequate if more than 70% of the BP measurements were recorded correctly.

Hypertension was defined on the basis of data from ABPM and according to the 2023 ESC/ESH Clinical Practice Guidelines for the Management of Arterial Hypertension. ²⁶ BP dipping was calculated by using the following formula: 1 - (average nighttime systolic BP / average daytime systolic BP). ²⁷ Values ranging from 10 to 20 percent were considered to be within normal limits. Patients with less than 10 percent BP dipping were categorized as non-dippers, while those with negative results (higher nighttime BP than daytime) were classified as risers. ^{26,27}

Potential Sources of Bias

The study utilized convenient sampling to recruit participants, which may not be representative of

all CKD patients and could lead to selection bias. Patients were assessed using a single 24-hour ABPM which may not be reproducible and result in measurement bias.

Confounding variables such as life style factors, comorbidities other than diabetes mellitus and medications taken by the patients were not evaluated due to the limited sample size.

Statistical Analysis

Quantitative variables, including age, BP measurements by ABPM and eGFR were described by using mean and standard deviation. Qualitative variables, such as sex, daytime and nighttime hypertension status, dipping pattern and diabetes mellitus were presented by using relative and absolute frequencies. The Chi-square test was used to compare variable frequencies between groups. To evaluate the averages of quantitative variables between two groups, the independent sample t-test or Mann-Whitney U-test was used for normally distributed and skewed variables, respectively. Analysis of variances was used to compare means between more than two groups. The relationship between continuous variables was assessed by using the Pearson correlation coefficient. Univariable and multivariable regression analyses were used to identify independent predictors of BP dipping for variables with an unadjusted P value of less than 0.2. All analyses were performed by using SPSS version 13 for Windows (SPSS Inc., IL, USA). A P value of less than 0.05 was considered significant.

RESULTS

The mean age of the patients was 56.34 years, and males comprised 41 cases (61.1%) of the sample. The mean levels of 24-hour systolic and diastolic BPs were found to be 128.15 and 75.81 mmHg, respectively (Table 1). Notably, a significant correlation was observed between daytime and nighttime systolic (Figure 1-a) and diastolic (Figure 1-b) BP measurements.

Approximately two-thirds of the study population (39 cases), including 28 males (71.8%) and 11 females (28.2%) had hypertension. Among these cases, 29 (46.8%) experienced daytime hypertension and 38 (61.3%) experienced nighttime hypertension. Additionally, 80.6% (50 cases) of the individuals had an abnormal pattern of BP dipping; out of which, thirty-two cases (51.6%) were classified

Table 1. Comparison of Demographic and ABPM Data Based on Dipping Pattern and Nighttime Hypertension Status in the Study Population

		BP dipping			Nighttime BP		
	Total	Dipper n = 12 (19.4%)	Non-dipper n = 50 (80.6%)	P	Normotensive n = 24 (38.7%)	Hypertensive n = 38 (61.3%)	P
Age, y	56.34 ± 12.9	55.33 ± 13.22	54.61 ± 12.71	.87	58.17 ± 12.05	55.39 ± 13.56	0.42
Sex							
Male (n (%))	41 (66.1%)	10 (83.3%)	31 (62 %)	.14	14 (58.3%)	27 (71.1%)	0.22
Female (n (%))	21 (33.9%)	2 (16.7%)	19 (38%)		10 (41.7%)	11 (28.9%)	
DM (n (%))	24 (38.7%)	1 (8.3%)	23 (46%)	.02	5 (20.8%)	19 (79.2%)	0.02
eGFR, mL/min/ 1.73m ²	34.66 ± 11.99	38.1 ± 12.87	35.79 ± 13.1	.6	36.27 ± 12.2	33.6 ± 12.05	0.4
CKD Stage (n (%))							
III-a	15 (24.2%)	4 (33.3%)	11 (22%)	.55	7 (29.2%)	8 (21.1%)	0.73
III-b	18 (29%)	4 (33.3%)	14 (28%)		7 (29.2%)	11 (28.9%)	
IV	29 (46.8%)	4 (33.3%)	25 (50%)		10 (41.7%)	19 (50%)	
24-hour SBP, mmHg	128.15 ± 19.48	116.46 ± 16.82	129.03 ± 17.5	.039	109.22 ± 7.79	139.51 ± 15.3	<0.005
24-hour DBP, mmHg	75.81 ± 10.99	73.75 ± 10.7	76.68 ± 9.43	.38	66.83 ± 5.04	81.32 ± 10.12	<0.005
Daytime SBP, mmHg	129.27 ± 18.74	121.25 ± 17.25	130.84 ± 17.35	.1	111.83 ± 9.23	139.76 ± 15.04	<0.005
Daytime DBP, mmHg	77.02 ± 11.03	77.25 ± 11.53	78.16 ± 9.46	.79	68.83 ± 6.27	82.08 ± 10.44	<0.005
Nighttime SBP, mmHg	124.8 ± 22.15	105.33 ± 15.84	123.71 ± 16.87	< .005	104.09 ± 8.2	137.34 ± 18.1	<0.005
Nighttime DBP, mmHg	72.49 ± 12.05	65.25 ± 9.36	72.45 ± 8.48	.02	62.57 ± 4.77	78.5 ± 11.12	<0.005
Daytime PP, mmHg	52.25 ± 13.04	44 ± 10.66	52.67 ± 12.54	.04	43 ± 5.79	57.68 ± 13.25	<0.005
Percent of dipping	3.56 ± 7.8	13.16 ± 2.76	5.43 ± 2.86	< .005	6.79 ± 1.41	7.85 ± 1.27	0.015
Non-dipping (n (%))	50 (80.6%)	_	_		16 (66.7%)	34 (89.5%)	0.03

Abbreviations: eGFR; estimated glomerular filtration rate, CKD; chronic kidney disease, SBP; systolic blood pressure, DBP; diastolic blood pressure, PP; pulse pressure

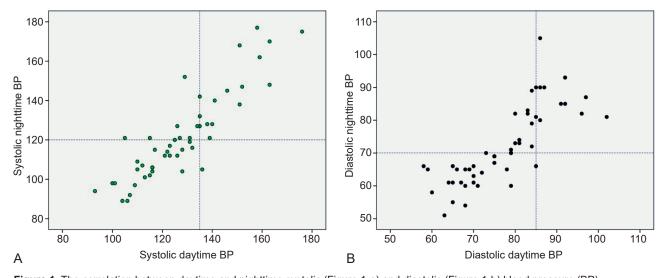


Figure 1. The correlation between daytime and nighttime systolic (Figure 1-a) and diastolic (Figure 1-b) blood pressure (BP) measurements. The vertical and horizontal lines represent the cutoff values for normal daytime and nighttime BP, respectively. Patients located in the upper left quadrants of the diagrams are affected by isolated nocturnal hypertension. On the other hand, patients in the lower right quadrant of the diagrams have daytime hypertension but normal nighttime BP levels.

as non-dippers, and eighteen cases (29%) were classified as risers. None of the study population exhibited an extreme pattern of dipping. Table 1 compares demographic and ABPM data based on dipping pattern and nighttime hypertension status. As shown, daytime pulse pressure was significantly higher in non-dippers (52.67 vs.

44 mmHg, P = .02), while systolic and diastolic daytime BP measurements did not significantly differ between normal dippers and those with abnormal BP dipping.

ABPM data revealed ten cases with isolated nighttime hypertension. Approximately 90% of patients with nighttime hypertension were non-

dippers. Moreover, all daytime BP measurements were significantly higher in individuals with nighttime hypertension, as shown in Table 1.

The mean eGFR in the study population was $34.66 \text{ mL/min} / 1.73\text{m}^2$. A significant correlation was found between the extent of BP dipping and the eGFR of the patients (R = 0.281, P = .02). The mean levels of nighttime BP dipping in patients with stage III-a, III-b and IV of CKD were 8.15, 2.46 and 1.84%, respectively with a statistically significant difference (P = .028) (Figure 2).

Twenty-four cases of the study population were affected by diabetes mellitus. The average amount of BP dipping was significantly lower in diabetic patients compared to non-diabetic cases (1.13 vs. 5.04%, P = .04). Additionally, diabetic patients were more likely to have nighttime hypertension, as shown in Table 1.

Table 2 shows the results of both univariable and

multivariable analyses, examining potential risk factors for predicting the amount of nighttime BP dipping. After adjusting for age, diabetes mellitus, and daytime pulse pressure in the multivariable model, it was found that eGFR was an independent factor in predicting the extent of BP dipping.

DISCUSSION

Abnormalities of nighttime BP, such as nighttime hypertension and non-dipping, are frequently overlooked when managing patients with chronic kidney diseases. However, these abnormalities can affect the cardiac and renal outcomes of CKD patients. Our research suggests that these abnormalities are prevalent among CKD patients, although the factors that predict the occurrence of each abnormality may vary.

Blunted BP dipping has been documented as an independent risk factor for CKD progression

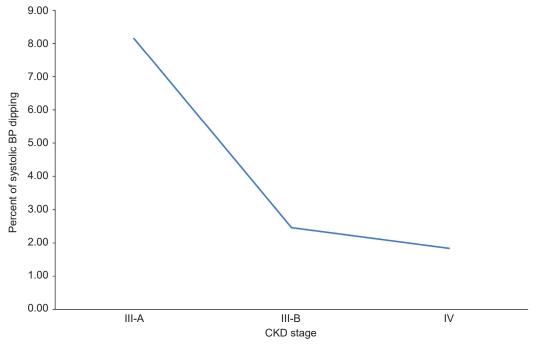


Figure 2. The mean level of systolic blood pressure dipping in different stages of chronic kidney disease.

Table 2. Univariable and Multivariable Regression Models for the Extent of Systolic Blood Pressure Dipping in the Study Population

	Univariable	Multivariable		
	B (95% CI)	P	B (95% CI)	P
Age	-0.123 (-0.276 to 0.031)	.115	-0.104 (-0.259 to 0.051)	.184
Diabetes Mellitus	-3.91 (-7.944 to 0.123)	.057	-2.109 (-7.332 to 3.114)	.422
Daytime PP	-0.167 (-0.316 to -0.019)	.028	-0.072 (-0.275 to 0.131)	.479
eGFR	0.182 (0.0200 to 0.343)	.028	0.171 (0.013 to 0.329)	.034

Abbreviations: PP; pulse pressure, eGFR; estimated glomerular filtration rate

and other target organ damage. 10,12 Several studies have evaluated predictors of blunted BP dipping in CKD patients. In a study by Mojon et al., CKD patients had higher ambulatory pulse pressure and significantly lower BP dipping.²⁸ Cha et al. found abnormal BP dipping in a majority of hypertensive CKD patients, but they did not find any relationship between eGFR and abnormal BP dipping patterns.²⁹ In the single study on Iranian CKD patients, it was found that 73.7% of these patients were nondippers. However, the authors did not discover any correlation between the circadian rhythm of BP and the severity of renal impairment. Although the study did not mention its inclusion criteria, the average serum creatinine level of the participants was 1.1 mg/dL, suggesting that most patients were in the early stages of CKD.¹⁹ Agarwal et al. reported that the extent of nocturnal BP dipping was associated with eGFR, serum albumin, younger age, and less proteinuria.30 Consistent with most previous studies, we found that a lower eGFR could be considered an independent predictor of impaired nighttime BP dipping.

Mechanisms such as increased sympathetic activity, volume overload, salt sensitivity, and sleep apnea have been proposed to be linked with kidney failure and impaired nighttime BP dipping.31-33 It is now believed that non-dipping may be a compensatory response to induce pressure natriuresis in volume overloaded CKD patients.³³ Consequently, several studies have shown that BP dipping is blunted in the early stages of kidney function impairment.34,35 Similar to the study by Agarwal et al., the prevalence of non-dipping in our study population was similar across the different stages of CKD.³⁵ However, our findings revealed that as CKD stage advances, the extent of BP dipping progressively decreases. This may reflect a greater contribution of underlying mechanisms such as volume overload, which leads to increased impairment in BP dipping.

Our results showed that BP dipping was significantly impaired in diabetic patients. However, after adjusting for other variables, diabetes mellitus could not be considered an independent predictor of nighttime BP dipping. In line with these findings, a study by Oh *et al.* revealed a significant correlation between the non-dipping pattern of BP and eGFR, regardless of the presence of diabetes mellitus.³⁶ Another study in diabetic

patients with CKD found that reduced eGFR was associated with a higher night-to-day systolic BP ratio.³⁷ In contrast to these results, Pistrosch *et al.* failed to find any relationship between eGFR and non-dipping in diabetic patients³⁸. Studies have shown a link between albuminuria, an indicator of early kidney injury, and blunted BP dipping in diabetic patients. Sommerfield *et al.* concluded that albuminuria is strongly associated with the non-dipping pattern of BP in diabetic patients.³⁹ Autonomic dysfunction, endothelial injury, and volume overload are proposed as potential underlying factors for these abnormalities in diabetic patients, which should be further investigated in future high-throughput studies.⁴⁰

Similar to the non-dipping pattern of BP in CKD patients, nighttime hypertension may also be an independent risk factor for adverse outcomes. The Dublin outcome study revealed that for every 10 mmHg increase in nighttime systolic BP, cardiovascular mortality increases by 21%.41 Consistent with previous research, we identified diabetes mellitus as a risk factor for nighttime hypertension.⁴² Additionally, we discovered a significant overlap between nighttime hypertension and the non-dipping pattern of BP, which may explain the high prevalence of nighttime hypertension in CKD patients.⁴³ Despite this overlap, our patients' nighttime BP was primarily influenced by their daytime BP levels and not associated with their eGFR. The distinct pathophysiological mechanism underlying nighttime hypertension remains unclear and requires further investigation in future research.

Our findings indicate that ABPM provides valuable data about nighttime BP abnormalities in CKD patients. However, the appropriate treatment for these abnormalities is not yet fully understood. Studies have revealed the importance of salt restriction in a better control of the nighttime BP and volume overload in patients with resistant hypertension.44 Additionally, most studies on antihypertensive chronotherapy show promising results in better managing nighttime BP and concomitant reducing of albuminuria in CKD patients. 45 Future high-throughput studies are necessary to better clarify the treatment modalities of nighttime BP abnormalities and evaluate the role of such treatments in improving the prognosis of CKD patients.

We encountered several limitations in this research. Firstly, our sample size was small. Secondly, we did not consider all factors that may influence nighttime BP patterns. For instance, we did not have access to data on antihypertensive medications. Additionally, we did not assess obstructive sleep apnea in our study population. Finally, we defined CKD based solely on eGFR and did not consider abnormalities in urinary sediment. Previous studies have shown a link between non-dipping and even slight proteinuria. Unfortunately, we did not have data on urinary protein excretion, preventing us from drawing any conclusions about the role of proteinuria in nighttime BP abnormalities in CKD patients.

CONCLUSION

Our results showed that nighttime BP abnormalities, including non-dipping and nighttime hypertension, are highly prevalent in patients with CKD. We found a significant overlap between nighttime hypertension and impaired BP dipping. We documented that eGFR is an independent predictor for the impaired BP dipping, and nighttime BP is mainly influenced by daytime BP measurements. Neither home nor office BP measurements were able to detect these abnormalities in nighttime BP. Therefore, our findings highlight the importance of ambulatory BP monitoring for better management of hypertension in CKD patients.

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