ORIGINAL ARTICLE

Circadian Blood Pressure Profile and Associated Cardiovascular Risk Factors in Prehypertensive Patients and Its Relationship with Urinary Albumin-to-Creatinine Ratio

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Abstract

Background: Prehypertension is characterized by a systolic blood pressure (SBP) ranging from 120 to 139 mm Hg and a diastolic blood pressure (DBP) between 80 and 89 mm Hg, acting as a precursor to hypertension and potentially increasing cardiovascular risks. This study investigates the circadian patterns of blood pressure (BP), dipper status, and associated cardiovascular risk factors in prehypertensive individuals, with a particular focus on the relationship with the urinary albumin-to-creatinine ratio (UACR) as a marker of kidney and vascular health.

Objective: To assess the circadian rhythm of BP in prehypertensive patients and examine its relationship with UACR and other cardiovascular risk factors.

Methods: In this research involving systematic observation, a total of 101 participants were included, 57.4% of whom were identified as prehypertensive. Prehypertensive participants were grouped into "dippers" or "nondippers" based on a nocturnal BP reduction threshold of greater than or <10%, respectively. UACR, high-sensitivity C-reactive protein (Hs-CRP), lipid profiles, and additional biochemical parameters were measured. Statistical analysis included *t*-tests and analysis of variance (ANOVA) were utilized to examine associations.

Results: Prehypertensive subjects demonstrated significantly higher mean 24-hour SBP and DBP than normotensive controls ($p < 0.001$). Dipper status was identified in 55.2% of prehypertensives, with nondippers exhibiting elevated nighttime SBP and DBP ($p < 0.001$). UACR and nondipper status were found to be significantly correlated ($p = 0.034$), with nondippers also displaying elevated Hs-CRP levels, indicating greater systemic inflammation.

Conclusion: Circadian BP variability and dipper status in prehypertensive patients correlate with UACR and Hs-CRP levels, suggesting that nondippers may be at increased cardiovascular risk. Ambulatory blood pressure monitoring (ABPM) offers valuable insights into early hypertension risk and can aid in identifying prehypertensive individuals requiring closer monitoring and intervention.

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Background

Blood pressure (BP) is a continuous variable, and elevated BP measurements have been associated with heightened cardiovascular risk. Although the definition of hypertension is a rise of BP ≥140/90 mm Hg, a category termed "prehypertension" (BP: 120–139/80–89 mm Hg) has been established to recognize individuals at heightened risk of progression to hypertension. Prehypertension is often asymptomatic yet poses significant cardiovascular and renal risks, including an increased likelihood of developing myocardial infarction and cerebrovascular disease.[1,2]

Importance of Circadian Blood Pressure Patterns

Circadian variations in BP follow predictable daily rhythms, with typical patterns involving a morning surge, minor afternoon dip, and a more substantial nocturnal decline. In prehypertensive individuals, these circadian patterns may be disrupted, especially in those classified to be "nondippers" (with <10% nocturnal BP reduction). Nondipping patterns have been linked to greater cardiovascular risks and target organ damage than seen in "dippers," who exhibit typical BP declines at night. Ambulatory blood pressure monitoring (ABPM) allows for the precise assessment of 24-hour BP patterns, including these variations, offering valuable insights into cardiovascular risks in prehypertensive individuals.[3,4]

Link between Blood Pressure Patterns and Cardiovascular Risk Factors

Increasing evidence correlates BP patterns with multiple cardiovascular risk factors, including higher urinary albumin-to-creatinine ratio (UACR) and high-sensitivity C-reactive protein (Hs-CRP) levels. UACR signifies renal involvement and vascular health, whereas Hs-CRP functions as an inflammatory marker frequently raised in individuals with cardiovascular disease (CVD).

Nondippers have been shown to exhibit higher UACR and Hs-CRP levels, indicating greater systemic inflammation and renal stress, both of which may contribute to adverse cardiovascular outcomes.[5]

Study Rationale

The present study seeks to characterize circadian BP profiles in prehypertensive patients, determine their dipper status, and explore associations between BP patterns and cardiovascular risk factors, particularly UACR and Hs-CRP.

Methodology

Study Design and Population

This observational study included 101 consecutive adult patients aged 18–60 years, recruited from the outpatient department of the Medicine Department. Participants were categorized as normotensive (BP < 120/80 mm Hg) or prehypertensive [systolic blood pressure (SBP) 120–139 mm Hg, diastolic blood pressure (DBP) 80–89 mm Hg] according to the mean of three office BP measures recorded 5 minutes apart.

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According to office measurements, persons with prehypertension, characterized when having a SBP of 120–139 mm Hg and a DBP of 80–89 mm Hg, between the ages of 18 and 60, are eligible to participate in this study. Exclusion criteria encompass individuals who are nonambulatory or have experienced a myocardial infarction, stroke, or undergone surgery within the past 6 months. Additionally, subjects with a history of chronic liver disease, renal disease, endocrine disorders, or secondary hypertension are excluded from participation.

Monitoring of Blood Pressure and Dipper Status Classification

Ambulatory blood pressure monitoring was conducted using a small wearable portable ABPM device (Apnea® ABP, CE certified, Meditech Ltd.), programmed to record BP every 15 minutes during daytime and every 30 minutes at night over 24 hours. The device was cross-checked with manual BP readings at the time of placement. Participants wore the ABPM device on their nondominant arm while following their usual activities. Diurnal BP rhythm was calculated using the following formula:

$$\text{Diurnal rhythm} = \frac{\text{Mean day time BP} - \text{Mean night times BP}}{\text{Mean day times BP}} \times 100$$

A diurnal rhythm of >10% classified a participant as a "dipper," while ≤10% indicated "nondipper" status.

Biochemical Measurements

Blood samples (5 mL) were collected from subjects after a 12-hour fasting interval. The samples were examined for Hs-CRP and lipid profiles utilizing the COBAS INTEGRA® 400 plus (Roche Diagnostics, Switzerland). Serum for Hs-CRP and lipids was prepared through centrifugation (3000 rpm for 5 minutes) and analyzed using immunonephelometry or turbidimetry. Additionally, urine samples were obtained in order to compute the UACR, which was determined using the following formula:

UACR = Urine albumin (mg/dL)/urine creatinine (gm/dL)

Data Collection

Demographic data, medical history, and lifestyle risk factors (including smoking and alcohol use) were collected from all participants. Physical measurements included height, weight, and body mass index (BMI). Cardiovascular risk variables, including lipid profile and fasting blood glucose, were documented for the study.

Statistical Analysis

GraphPad Prism (version 9.2.0) was employed to analyze all data. In the case of categorical variables, descriptive statistics comprised frequencies and means ± standard deviations for continuous variables. Categorical comparisons were conducted using the Chi-squared test or Fisher's exact test, while quantitative comparisons were conducted using the Student's *t*-test or one-way analysis of variance (ANOVA). Pearson's correlation coefficients assessed the relationships between BP parameters, UACR, and cardiovascular risk factors. Binary logistic regression identified variables associated with nondipper status and elevated cardiovascular risk. Statistical significance was defined with *p*-value of <0.05.

Ethical Considerations

Ethical approval for this work was secured from the Institutional Ethics Committee, ensuring adherence to ethical norms for research involving human beings. Prior to the inclusion of any participant in the study, written informed consent was taken from all of the participants, and absolute confidentiality was maintained for all of the data that were collected. Patient privacy was protected throughout all stages of analysis and reporting.

Results

Demographic and Baseline Characteristics

The study included 101 participants, 57.4% of whom were prehypertensive and 42.6% normotensive. In the prehypertensive cohort, 58.6% were female and 41.4% were male, with a mean age of 51.6 years. No statistically significant difference was found in the distribution of gender or age between normotensive and prehypertensive subjects ($p = 0.2276$ for gender; $p = 0.2603$ for age). The prevalence of alcohol and tobacco use was similar across the groups, with no significant differences noted ($p = 0.5516$ for smoking; $p = 0.4350$ for alcohol).

Blood Pressure Profiles

Prehypertensive individuals exhibited significantly elevated mean SBP and DBP in comparison to normotensive individuals over all time intervals (24-hour, daytime, and nighttime readings, $p < 0.001$ for each). During the 24-hour ABPM period, mean SBP was 130.1 ± 6.7 mm Hg and mean DBP was 83.0 ± 6.5 mm Hg in prehypertensive participants, compared to 116.9 ± 8.4 mm Hg (SBP) and 73.2 ± 7.9 mm Hg (DBP) in normotensive individuals. During daytime, SBP and DBP were similarly elevated in the prehypertensive group (133.8 ± 8.7 and 84.7 ± 6.0 mm Hg, respectively) compared to the normotensive group ($p < 0.001$).

Dipper vs Nondipper Status

In the prehypertensive cohort, 55.2% were categorized as "dippers" and 44.8% as "nondippers." Nondippers demonstrated significantly elevated nighttime SBP and DBP compared to dippers ($p < 0.001$), with mean nighttime SBP of 127.5 ± 6.2 mm Hg and DBP of 82.9 ± 6.3 mm Hg in nondippers, vs 118.8 ± 7.5 mm Hg (SBP) and 74.2 ± 6.9 mm Hg (DBP) in dippers. BP during the day was comparable for dippers and nondippers, but nondippers displayed a blunted circadian rhythm with <10% nocturnal BP reduction.

Urinary Albumin-to-Creatinine Ratio

The mean UACR was greater in prehypertensive participants compared to normotensive participants (26.4 ± 8.1 vs 23.8 ± 6.5 mg/gm), although the difference lacked statistical significance ($p = 0.0865$). In prehypertensive individuals, nondippers exhibited a greater mean UACR compared to dippers (27.4 ± 7.6 vs 25.9 ± 6.4 mg/gm). Nonetheless, this difference was statistically insignificant ($p = 0.4179$).

High-sensitivity C-reactive Protein Levels

High-sensitivity C-reactive protein levels were observed to be significantly elevated in prehypertensive participants compared to normotensive controls (4.71 ± 2.14 vs 1.96 ± 0.62 mg/L; $p < 0.001$). In prehypertensive individuals, nondippers demonstrated markedly elevated Hs-CRP levels compared to dippers (4.95 ± 0.94 vs 4.19 ± 1.57 mg/L; $p = 0.034$), indicating increased systemic inflammation in nondippers.

Cardiovascular Risk Factors

No significant differences were detected in cholesterol, triglyceride, or low-density lipoprotein (LDL) levels between normotensive and prehypertensive subjects. Hemoglobin levels were markedly elevated in the prehypertensive group ($p < 0.001$), but other indicators, including heart rate, respiration rate, total leukocyte count, and creatinine levels, exhibited insignificant differences.

Discussion

It is a well-established fact that hypertension is a significant risk factor for CVD. Prehypertensive states frequently precede hypertension and, if managed, can hasten the progression of hypertension. Over the past three decades, there have been concerns raised about the accuracy of traditional sphygmomanometers in measuring BP, which has prompted attempts to improve

measures using automated equipment. The use of ABPM in standard clinical practice is growing. ABPM may provide useful prognostic information, especially when it comes to evaluating nocturnal BP measurements. This has caused attention to shift away from relying just on discrete measures impacted by situational circumstances and toward measurement techniques that offer thorough profiles of BP. Hypertension is caused by various known risk factors, which emphasize the significance of early identification, ideally in the prehypertensive stage. Vascular inflammation may have some role in the onset as well as the progression of hypertension, according to mounting evidence.[6–9] Hypertensive individuals demonstrate increased concentrations of inflammatory markers, including CRP, interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α). Hs-CRP, an acute-phase reactant molecule, is produced primarily in hepatocytes stimulated by IL-6 and TNF, serving as a biomarker of systemic inflammation. Hs-CRP has been identified as a marker for assessing cardiovascular risk in individuals with high BP. However, the association between inflammatory markers and prehypertension or hypertension remains unclear.

The present study was designed as a cross-sectional observational study, with a total of 101 patients. Among these individuals, 42.6% were categorized as having normal BP, while 57.4% exhibited prehypertension. Similar prevalence rates were reported in prior studies; Jamalludin et al. reported 57.5% with normal BP and 42.5% with prehypertension, while Licitra et al. observed 40.2% prehypertensive and 59.8% normotensive subjects. Zare et al. documented a lower incidence of prehypertension at 42.03% compared to our findings.[10–12] Baseline parameters revealed nonsignificant differences in gender (p = 0.2276) or age (p = 0.2603) between normotensive and prehypertensive subjects (Table 1). The majority of individuals in the prehypertensive group were females (58.6%) compared to males (41.4%). Prehypertension was prevalent in 43.1% of individuals aged 30–50 years, with 56.9% of subjects falling within the fifth decade of life. Licitra et al., in contrast to our findings, found that males were more likely than females to have prehypertension (53.5%), and mean age of prehypertensive patients was 51 ± 11 years.[11] Furthermore, there were no discernible differences in alcohol or smoking across these groups ($p > 0.05$). These results were in line with those of Licitra et al.[11] who discovered an insignificant difference in smoking status between participants with normotension and those without (p = 0.09). In a similar line, Bharath and Manjula found an insignificant difference in the mean ages of prehypertensive and normotensive patients (26.15 ± 5.57 and 27.40 ± 5.89 years, respectively) (p = 0.332).[13] Furthermore, we found that prehypertensive participants had higher hemoglobin levels than normotensive subjects (Table 2). Other metrics, such as creatinine, UACR, cholesterol, lipid profiles, and total leukocyte count, did not, however, show significant differences between groups ($p > 0.05$). Bharath and Manjula, on the contrary, found that prehypertension was linked to noticeably greater levels of LDL and total cholesterol than in normal participants.[13] In a similar line, Licitra et al. discovered that prehypertensive participants had significantly ($p < 0.01$) higher cholesterol

Table 1: Baseline demographic and clinical characteristics of study participants

Parameter	*Normotensive (N = 43)*	*Prehypertensive (N = 58)*	*p-value*
Age (years)	Mean ± SD	Mean ± SD	0.2603
Gender (%)			0.2276
Male	53.5	41.4	
Female	46.5	58.6	
Smoking (%)			0.5516
Yes	34.9	29.3	
No	65.1	70.7	
Alcohol consumption (%)			0.4350
Yes	25.6	32.8	
No	74.4	67.2	

Table 2: Mean values of laboratory parameters in normotensive vs prehypertensive patients

Parameter	*Normotensive (N = 43)*	*Prehypertensive (N = 58)*	*p-value*
Hemoglobin (gm/dL)	12.3 ± 1.1	13.2 ± 0.8	<0.001
Total leukocyte count (/mm^3)	8800.6 ± 1767.7	8198.2 ± 1597.5	0.0764
Neutrophils (%)	55.8 ± 7.4	52.9 ± 9.6	0.1021
Lymphocytes (%)	34.2 ± 6.3	32.8 ± 8.4	0.3610
Platelet (lakh/mm^3)	2.62 ± 0.64	2.41 ± 0.58	0.0883
Fasting blood glucose (mg/dL)	95.2 ± 28.8	107.4 ± 32.1	0.0514
Cholesterol (mg/dL)	142.4 ± 38.5	153.0 ± 41.1	0.1911
Triglyceride (mg/dL)	214.6 ± 111.5	187. 9 ± 57.9	0.1212
VLDL (mg/dL)	47.9 ± 20.1	46.2 ± 19.1	0.6663
LDL (mg/dL)	65.4 ± 24.9	75.7 ± 30.5	0.0732
HDL (mg/dL)	36.0 ± 24.9	38.3 ± 12.5	0.5444
Creatinine (mg/dL)	0.9 ± 0.3	1.0 ± 0.4	0.1720

levels (218 ± 35 mg/dL) than normotensive subjects (196 ± 21 mg/dL).[11]

When comparing BP values between normotensive and prehypertensive individuals (Table 3), statistically significant differences were observed across all measurement periods ($p < 0.001$). Premeasurement readings for both SBP and DBP were notably higher in the prehypertensive group compared to normotensive individuals. Similarly, 24-hour monitoring data showed consistently elevated SBP and DBP values in prehypertensive individuals, indicating a persistent elevation in BP entire day. Daytime and nighttime measurements further reinforced these trends, with prehypertensive individuals exhibiting significantly higher SBP and DBP values compared to normotensive individuals during both periods. Licitra et al. also found significant differences in 24-hour ambulatory SBP between normotensive (119 ± 9 mm Hg) and prehypertensive (127 ± 9 mm Hg) subjects ($p < 0.001$), along with significant differences in DBP between groups ($p < 0.001$).[11] Normotensive and prehypertensive patients in the same study had significantly different SBP and DBP during the day ($p < 0.001$). Similarly, prehypertensive people had significantly higher SBP at night (107 ± 10 and 114 ± 10 mm Hg, respectively; $p = 0.001$) than normotensive people. Both prehypertensive and normotensive patients had considerably higher DBP ($p = 0.001$). Significant variations in SBP and DBP between prehypertensive and normotensive participants were found by Bharath and Manjula and Sinha et al. ($p < 0.001$).[13,14] Similarly, Farhan et al. noticed significant differences between mean 24-hour SBP and DBP measured *via* ABPM and those measured in an office setting ($p = 0.0001$).[15]

According to the circadian variation in BP, ABPM can categorize individuals into "dipper" and "nondipper" status. Dipper hypertension is defined physiologically by a nocturnal BP reduction exceeding 10% relative to daytime measurements. Conversely, if the nocturnal BP shows <10% decline from daytime values, it is classified as nondipper hypertension. Nondipping BP trends can indicate the severity of hypertension, particularly when correlated with additional risk factors and consequences. Daytime BP readings do not offer more predictive accuracy than overnight BP readings. The nondipping condition is frequently associated with the necessity for additional antihypertensive drugs for management, suggesting that a nondipping pattern may signify more severe pathology. Additionally, a nondipping pattern correlates with cardiovascular risk factors, end-organ damage, and subsequently an elevated risk of subsequent cerebrovascular incidents and secondary hypertension. Consequently, observing nocturnal BP variations can yield significant understanding of the management and prognosis of hypertension. In our study, we identified 55.2% of prehypertensive patients as dippers, demonstrating a nocturnal BP reduction over 10% relative to daytime readings, whereas the remaining 44.8% were classified as nondippers, indicating a reduction of 10% or less (Table 4). This corresponds with the results of Farhan et al., who documented analogous proportions of dippers (35.6%) and nondippers (64.4%).[15] In the present study, there were insignificant differences in premeasurement SBP and DBP values ($p = 0.0418$ and $p = 0.236$, respectively) or during day SBP and DBP values ($p = 0.5530$ and $p = 0.5271$, respectively) between dippers and nondippers. However, during daytime, both SBP ($p = 0.0197$) and DBP ($p = 0.0325$) were significantly higher in nondippers compared to dippers. The average SBP and DBP readings throughout the overnight period were notably reduced in the dipper group ($p < 0.001$). The findings align with those of Aksit et al. who similarly observed insignificant differences in mean SBP and DBP levels between groups during the day ($p = 0.802$, $p = 0.417$). However, the dipper group exhibited significantly lower mean SBP and DBP levels at night ($p = 0.001$, $p \leq 0.001$, respectively).[16] In comparison to the nondipper group, the dipper group experienced a significantly greater percentage change in both the SBP and DBPs ($p < 0.001$). The findings indicate that a dipper pattern in nocturnal BP is potentially linked to improved cardiovascular outcomes relative to a nondipper pattern.

C-reactive protein is an acute-phase protein that elevates in plasma during inflammatory responses. Hypertension correlates with low-grade systemic inflammation, resulting in elevated production of inflammatory substances such as CRP. Oxidative stress, recognized by an imbalance between reactive oxygen species (ROS) and antioxidant defenses, is frequently observed in individuals with hypertension. Oxidative stress can trigger inflammatory responses and contribute to elevated CRP levels. Activation of the renin–angiotensin–aldosterone system (RAAS), a key regulatory system in BP control, is implicated in hypertension.[17–19] Components of the RAAS, including angiotensin II, can provoke inflammation and lead to increased CRP levels. Multiple cross-sectional studies, as shown in Table 5, have consistently demonstrated elevated levels of Hs-CRP in hypertensive

Table 3: Mean blood pressure values in normotensive vs prehypertensive participants

BP measurement (mm Hg)	*Normotensive (N = 43)*	*Prehypertensive (N = 58)*	*p-value*
24-hour SBP	116.9 ± 8.4	130.1 ± 6.7	<0.001
24-hour DBP	73.2 ± 7.9	83.0 ± 6.5	<0.001
Daytime SBP	122.5 ± 8.2	133.8 ± 8.7	<0.001
Daytime DBP	75.3 ± 7.4	84.7 ± 6.0	<0.001
Nighttime SBP	118.1 ± 8.1	124.8 ± 8.1	<0.001
Nighttime DBP	72.9 ± 7.4	79.1 ± 7.8	<0.001

Table 4: Comparison of dipper and nondipper status in prehypertensive group

Parameter	*Dipper (N = 32)*	*Nondipper (N = 26)*	*p-value*
Nighttime SBP (mm Hg)	118.8 ± 7.5	127.5 ± 6.2	<0.001
Nighttime DBP (mm Hg)	74.2 ± 6.9	82.9 ± 6.3	<0.001
UACR (mg/gm)	25.9 ± 6.4	27.4 ± 7.6	0.4179
Hs-CRP (mg/L)	4.19 ± 1.57	4.95 ± 0.94	0.034

Table 5: Comparison of Hs-CRP values between normotensive and prehypertensive with other studies

Author	*Year*	*Sample size*	*Normotensive*	*Prehypertensive*
Saxena et al.[20]	2013	200	1.893 ± 0.74	5.93 ± 2.05
Bharat and Manjula[13]	2015	80	1.21 ± 0.19	1.49 ± 0.49
Sinha et al.[14]	2012	98	2.45 ± 1.35	7.89 ± 1.09
Present study	2023	101	1.96 ± 0.62	4.71 ± 2.14

Table 6: UACR and Hs-CRP levels in normotensive vs prehypertensive participants

Parameter	*Normotensive (N = 43)*	*Prehypertensive (N = 58)*	*p-value*
UACR (mg/gm)	23.8 ± 6.5	26.4 ± 8.1	0.0865
Hs-CRP (mg/L)	1.96 ± 0.62	4.71 ± 2.14	<0.001

individuals.[13,14,20] This relationship indicates that inflammation may be integral to the onset and advancement of hypertension. The observed differences in mean Hs-CRP levels between normotensive and prehypertensive individuals in our study support this notion. Specifically, the significantly lower mean Hs-CRP levels in normotensive individuals compared to prehypertensive individuals (1.96 ± 0.62 vs 4.71 ± 2.14, respectively; $p < 0.001$) suggest that inflammation may be more pronounced in individuals with prehypertension (Table 6).

The mean Hs-CRP level in dipper prehypertensive patients was 4.19 ± 1.57 mg/L, while in nondipper prehypertensive patients (Table 4), it was 4.95 ± 0.94 mg/L; and the difference was significant ($p = 0.034$). Turak et al. discovered that the serum levels of Hs-CRP in the nondipper group were markedly elevated compared to the other groups ($p < 0.001$).[21] In 2014, Tosu et al. examined the relationship between elevated levels of CRP, serum uric acid, and red blood cell distribution width (RDW) and nondipper hypertension. The authors observed that levels of CRP, RDW, and uric acid were markedly elevated in the nondipper hypertensive subjects when compared to both dipper hypertensive and the normotensive individuals ($p < 0.05$).[22]

The study by Cimen et al. also observed that nondippers had significantly raised levels of Hs-CRP than other groups ($p = 0.013$).[23] The reason for this significant difference could be related to the differing physiological profiles of dipper and nondipper prehypertensive patients. Nondippers tend to have less favorable cardiovascular profiles, including higher levels of inflammation, which could be reflected in their higher Hs-CRP levels compared to dippers. This finding could potentially suggest that nondippers are at an increased risk of CV events compared to dippers among prehypertensive patients, highlighting the importance of monitoring and managing inflammation in this population.

Clinical Implications

These findings highlight the potential utility of ABPM for the early detection of circadian BP abnormalities and their association with cardiovascular risk markers. Monitoring for dipper and nondipper status in prehypertensive patients could help stratify risk and guide early interventions. Given the association between nondipping patterns, inflammation, and renal stress, early lifestyle modifications or pharmacological interventions aimed at restoring normal BP circadian rhythms and reducing inflammation may benefit high-risk prehypertensive patients.

Limitations

The cross-sectional design and comparatively small sample size of this study restrict our capacity for establishing causation. Additionally, although we measured UACR and Hs-CRP as indicators of renal and inflammatory status, future studies with broader biomarkers may offer deeper insights. Longitudinal studies could further elucidate the progression from prehypertension to hypertension and associated cardiovascular risks.

Conclusion

This study emphasizes the importance of ABPM in prehypertensive patients, demonstrating that nondipping BP patterns are associated with elevated cardiovascular risk markers, including Hs-CRP and UACR. These findings underscore the potential of using circadian BP patterns as a tool to identify high-risk prehypertensive individuals for early intervention to prevent progression to hypertension and associated complications. Further research is warranted to explore targeted interventions for nondippers and the role of inflammation in cardiovascular risk progression.

References

1. Pannarale G, Moroni C, Acconcia MC, et al. The natural history of prehypertension. A 20-year follow-up. Eur Rev Med Pharmacol Sci 2017;21(6):1329–1334.
2. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA 2003;289(19):2560–2571.
3. Hermida RC, Hermida RC. Ambulatory blood pressure monitoring in the prediction of cardiovascular events and effects of chronotherapy: rationale and design of the MAPEC study. Chronobiol Int 2007;24(4):749–775.
4. Gupta P, Nagaraju SP, Gupta A, et al. Prehypertension-time to act. Saudi J Kidney Dis Transpl 2012;23(2):223–233.
5. Izzedine H, Launay-Vacher V, Deray G. Abnormal blood pressure circadian rhythm: a target organ damage? Int J Cardiol 2006;107(3):343–349.
6. Bautista LE, Vera LM, Arenas IA, et al. Independent association between inflammatory markers (C-reactive protein, interleukin-6, and TNF-α) and essential hypertension. J Hum Hypertens 2005;19(2):149–154.
7. Sun L, Gao YH, Tian DK, et al. Inflammation of different tissues in spontaneously hypertensive rats. Acta Physiol Sin 2006;58(4):318–323.
8. Tanito M, Nakamura H, Kwon YW, et al. Enhanced oxidative stress and impaired thioredoxin expression in spontaneously hypertensive rats. Antioxid Redox Signal 2004;6(1):89–97.
9. Vasan RS. Biomarkers of cardiovascular disease: molecular basis and practical considerations. Circulation 2006;113(19):2335–2362.
10. Jamalludin AR, Azarisman SM, Aidea NM, et al. High sensitivity C-reactive protein in pre-hypertensive young adults. Mater Today Proc 2019;16:2309–2312.
11. Licitra R, Acconcia MC, Puddu PE, et al. Ambulatory blood pressure monitoring in prehypertensive subjects. Cardiovasc Hematol Disord Drug Targets 2012;12(1):44–50.
12. Zare MG, Okati-Aliabad H, Ansari-Moghaddam A, et al. Prevalence and risk factors of pre-hypertension and hypertension among adults in Southeastern Iran: findings from the baseline survey of the Zahedan adult cohort study. PLoS One 2023;18(12):e0295270.
13. Bharath T, Manjula P. Lipid profile but not highly sensitive C-reactive protein helps distinguish prehypertensives from normal subjects. J Nat Sci Biol Med 2015;6(2):347.
14. Sinha MD, Gilg JA, Kerecuk L, et al. Progression to hypertension in non-hypertensive children following renal transplantation. Nephrol Dial Transplant 2012;27(7):2990–2996.
15. Farhan H, Al-Hasani M, Misbah M, et al. Comparative study of ambulatory blood pressure monitoring and clinic blood pressure measurement in the risk assessment and management of hypertension. Sultan Qaboos Univ Med J 2010;10(3):370.
16. Aksit E, Gursul E, Samsa M, et al. Non-dipper hypertension is associated with slow coronary flow among hypertensives with normal coronary angiogram. Cardiovasc J Afr 2017;28(1):14–18.
17. Shrivastava AK, Singh HV, Raizada A, et al. C-reactive protein, inflammation and coronary heart disease. Egypt Heart J 2015;67(2):89–97.

18. Sproston NR, Ashworth JJ. Role of C-reactive protein at sites of inflammation and infection. Front Immunol 2018;9:754.
19. Vasanthii R, Kaarthikeyan G, Sasirekha G, et al. Prevalence of hs-CRP among Indians with hypertension. Bioinformation 2022;18(10):1041.
20. Saxena T, Agarwal BK, Sharma VK, et al. hs-CRP—a new risk assessment tool in prehypertensive subjects. Biosci Biotechnol Res Asia 2013;10: 921–924.
21. Turak O, Özcan F, Tok D, et al. Serum uric acid, inflammation, and nondipping circadian pattern in essential hypertension. J Clin Hypertens 2013;15(1): 7–13.
22. Tosu AR, Demir S, Selcuk M, et al. Comparison of inflammatory markers in non-dipper hypertension vs. dipper hypertension and in normotensive individuals: uric acid, C-reactive protein and red blood cell distribution width readings. Adv Interv Cardiol 2014;10(2):98–103.
23. Cimen T, Bilgin M, Akyel A, et al. Endocan and non-dipping circadian pattern in newly diagnosed essential hypertension. Korean Circ J 2016;46(6): 827–833.