

Original Research Article

ASSOCIATION OF AMBULATORY ARTERIAL STIFFNESS INDEX (AASI) AND NOCTURNAL BLOOD PRESSURE DIPPING PATTERNS IN NORMOTENSIVE YOUNG ADULT MALES

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ABSTRACT

Background: Blood pressure follows a circadian variation. The decrease in BP during sleep is referred to as “nocturnal dipping” and is partly attributable to decrease in sympathetic output. Diminished nocturnal dipping of BP is a strong, independent predictor of hypertension. Ambulatory arterial stiffness index (AASI) is a marker of arterial stiffness and a well known predictor of cardiovascular mortality. This study was intended to investigate the association between AASI and dipping status in normotensive young adult males. The objective is to assess dipping state in healthy young adult males using 24 hours ambulatory blood pressure. To assess AASI in healthy young adult males using 24 hours ambulatory blood pressure. To compare AASI in dippers, non – dippers and reverse dippers.

Materials and Methods: The study included 60 healthy young adults of age between 20 to 35 years. Their dipper state was assessed by 24 hours Ambulatory blood pressure measurement done by Contec Ambulatory Blood Pressure Monitor and they were classified as dippers, non dippers and reverse dippers. AASI was calculated by the formula one minus regression score of diastolic over systolic BP. AASI scores were compared among dippers, non-dippers and reverse dippers.

Results: The mean age of subjects was 24.36 ± 4.58 years. Out of the 60 subjects, 19 were found to be non-dippers and none was found to be reverse dipper. The mean AASI score of non-dippers was significantly higher than dippers [0.39 ± 0.26 vs 0.26 ± 0.09 ; $p < 0.01$].

Conclusion: The present study indicated that non-dipping patterns may be associated with increased arterial stiffness and hence increased cardiovascular morbidity.

Keywords: 24 hour ambulatory BP monitoring, Ambulatory arterial stiffness index, Dipping pattern.

INTRODUCTION

Cardiovascular diseases (CVDs) remain the leading cause of morbidity and mortality worldwide. Traditionally, cardiovascular risk assessment has focused on office blood pressure measurements; however, increasing evidence suggests that ambulatory blood pressure monitoring (ABPM) provides superior prognostic information regarding

cardiovascular outcomes. Blood pressure is not a static physiological parameter but follows a circadian rhythm characterized by higher values during daytime activity and a physiological decline during sleep. This nocturnal reduction in blood pressure is commonly referred to as “nocturnal dipping.”^[1]

In healthy individuals, nocturnal systolic and diastolic blood pressures decrease by approximately 10–20% compared to daytime values. This reduction

is primarily mediated by decreased sympathetic nervous system activity, enhanced vagal tone, reduced physical activity, and changes in hormonal secretion during sleep.^[2] Individuals demonstrating a nocturnal decline of at least 10% are classified as "dippers," whereas those with a decline of less than 10% are termed "non-dippers." In some individuals, blood pressure may paradoxically rise during sleep, a phenomenon known as "reverse dipping."^[3] Growing evidence indicates that non-dipping and reverse-dipping patterns are associated with adverse cardiovascular outcomes independent of mean blood pressure levels. Non-dipping status has been linked with left ventricular hypertrophy, endothelial dysfunction, carotid intima-media thickening, chronic kidney disease, and future development of hypertension.^[4,5] Importantly, these abnormalities may be present even in apparently normotensive individuals, suggesting that alterations in circadian blood pressure regulation may represent an early marker of cardiovascular risk.

Arterial stiffness is another important predictor of cardiovascular morbidity and mortality. Stiffening of large arteries results in increased systolic blood pressure, widened pulse pressure, increased cardiac workload, and impaired coronary perfusion. Traditionally, pulse wave velocity has been considered the gold standard measure of arterial stiffness. However, its assessment requires specialized equipment and expertise, limiting its routine use in clinical practice.^[6]

The Ambulatory Arterial Stiffness Index (AASI) is a simple and non-invasive parameter derived from 24-hour ABPM recordings. It is calculated as one minus the regression slope of diastolic blood pressure on systolic blood pressure obtained from ambulatory recordings. Higher AASI values indicate greater arterial stiffness and have been associated with cardiovascular mortality, stroke, target-organ damage, and future hypertension.^[7]

Several studies in hypertensive and elderly populations have demonstrated an association between increased arterial stiffness and abnormal nocturnal blood pressure patterns.^[8] However, limited data are available regarding this relationship in healthy young adults. Young adulthood represents a critical period during which subclinical cardiovascular alterations may emerge long before overt hypertension develops. Identification of early vascular changes in this population may facilitate timely preventive interventions.

Understanding whether non-dipping status is associated with increased arterial stiffness in normotensive young adults could provide valuable insights into the early pathogenesis of cardiovascular disease. Therefore, the present study was undertaken in healthy young adult males to assess nocturnal blood pressure dipping status using 24-hour ambulatory blood pressure monitoring and to determine the Ambulatory Arterial Stiffness Index (AASI) from the same recordings. The study further aimed to compare AASI values among individuals

classified as dippers, non-dippers, and reverse dippers, thereby exploring whether differences in nocturnal blood pressure patterns are associated with variations in arterial stiffness in this apparently healthy population.

MATERIALS AND METHODS

Study Design and Setting: The present study was conducted as a cross-sectional observational study among healthy normotensive young adult males. The study was carried out in the Department of Physiology of a tertiary care teaching institution over a period of 12 months, following approval from the Institutional Ethics Committee. All participants were informed about the study procedures, and written informed consent was obtained before enrolment.

Study Population: The study population comprised healthy male volunteers aged 20–35 years who were recruited from students, staff members, and healthy attendants associated with the institution. Participants were considered eligible if they were healthy males within the specified age range, had a clinic blood pressure below 140/90 mmHg, and were willing to participate and provide written informed consent.

Individuals with a known history of hypertension, cardiovascular disease, renal disease, diabetes mellitus, or endocrine disorders known to affect blood pressure were excluded. Smokers, chronic alcohol users, shift workers, and individuals with diagnosed sleep disorders were also excluded because these factors may influence circadian blood pressure patterns. Participants with incomplete or inadequate 24-hour ambulatory blood pressure monitoring (ABPM) recordings were excluded from the final analysis.

Sample Size: The study was planned as a pilot exploratory study. A convenient sample of 60 eligible male participants was recruited. The sample size was determined primarily on the basis of study feasibility and supported by previous studies evaluating nocturnal blood pressure dipping patterns and arterial stiffness in healthy populations. The sample was considered adequate for preliminary exploration of the relationship between AASI and nocturnal blood pressure dipping status and for comparison of AASI among the different dipping patterns.

Procedure for Collection of Data: After obtaining informed written consent, demographic information and relevant medical history were recorded. Physical examination and clinic blood pressure measurements were performed to confirm eligibility.

Twenty-four-hour ambulatory blood pressure monitoring was conducted using a Contec Ambulatory Blood Pressure Monitor. Participants were instructed to continue their routine daily activities while avoiding strenuous exercise during the monitoring period.

The ABPM device was programmed to record blood pressure at an interval of 15 minutes while awake and 30 minutes while sleeping throughout the 24-hour

period. Participants maintained a diary documenting sleep and wake times. Daytime and nighttime periods were identified according to individual sleep schedules.

Assessment of Dipping Status: Nocturnal blood pressure dipping percentage was calculated using the formula:

$$\text{Dipping (\%)} = \frac{[(\text{Mean Daytime SBP} - \text{Mean Nighttime SBP}) / \text{Mean Daytime SBP}] \times 100}{1}$$

Participants were classified as:

- Dippers: nocturnal BP reduction $\geq 10\%$
- Non-dippers: nocturnal BP reduction $< 10\%$
- Reverse dippers: nighttime BP greater than daytime BP

Assessment of Ambulatory Arterial Stiffness Index: AASI was calculated according to the standard formula:

$$\text{AASI} = 1 - \text{Regression slope of DBP on SBP}$$

The regression slope was derived from all ambulatory systolic and diastolic blood pressure readings obtained during the monitoring period.

Higher AASI values were considered indicative of increased arterial stiffness.

Statistical Analysis: Data were entered into Microsoft Excel and analysed using SPSS software version 25.0.

Continuous variables were expressed as mean $\pm$ standard deviation (SD). Categorical variables were expressed as frequencies and percentages.

Comparison of AASI values between dippers and non-dippers was performed using the independent samples Student's t-test. Statistical significance was considered at $p < 0.05$.

RESULTS

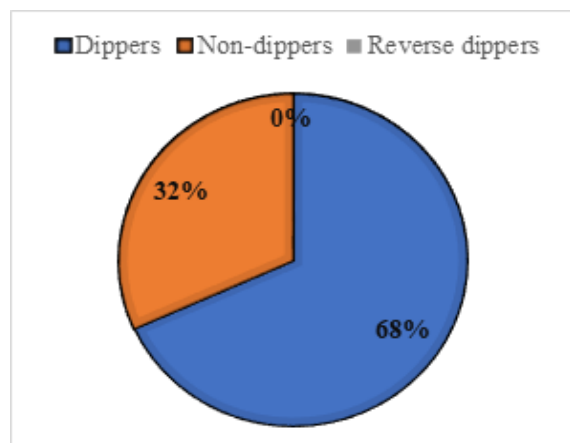


Figure 1: Distribution of participants according to dipping status (%)

A total of 60 healthy normotensive young adult males were included in the study. The mean age of the participants was 24.36 ± 4.58 years. Based on the nocturnal blood pressure profile obtained from 24-hour ambulatory blood pressure monitoring, as shown in [Figure 1], 41 participants (68.3%) were classified as dippers and 19 (31.7%) as non-dippers, while none demonstrated a reverse-dipping pattern.

Thus, nearly one-third of the study participants showed an altered nocturnal blood pressure dipping pattern despite being apparently healthy and normotensive.

The mean AASI was 0.26 ± 0.09 among dippers and 0.39 ± 0.26 among non-dippers. AASI was significantly higher in non-dippers than in dippers ($p < 0.01$), indicating a difference in arterial stiffness between the two nocturnal blood pressure phenotypes [Figure 2]. As no participant exhibited a reverse-dipping pattern, comparison of AASI with this subgroup was not applicable.

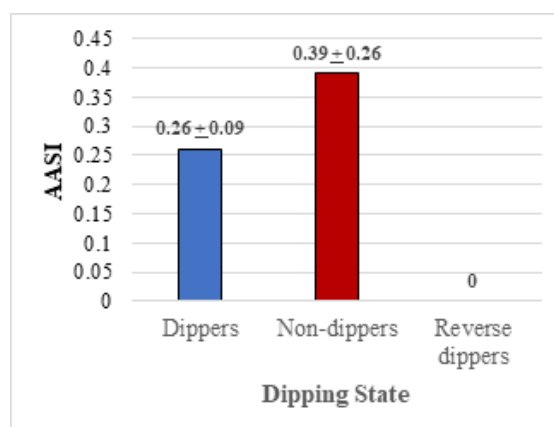


Figure 2: Comparison of AASI with Dipping status

DISCUSSION

The present study investigated the relationship between nocturnal blood pressure dipping patterns and ambulatory arterial stiffness index in healthy normotensive young adult males. The findings indicate that non-dipping status is associated with significantly higher AASI values, suggesting the presence of increased arterial stiffness despite normal clinic blood pressure levels.

The mean age of participants in the present study was 24.36 years, representing a relatively young and healthy population. Despite their normotensive status, about one third of the participants were classified as non-dippers. This observation is clinically important because abnormal circadian blood pressure variation has been increasingly recognized as an early marker of cardiovascular risk.^[9]

The prevalence of non-dipping observed in the present study is comparable to that reported in previous investigations involving young adults and healthy populations. Hermida et al. reported that a substantial proportion of apparently healthy individuals may demonstrate attenuated nocturnal blood pressure decline despite normal daytime blood pressure values.^[10] Similarly, Cuspidi et al. observed that non-dipping status may occur even in normotensive subjects and is associated with subclinical cardiovascular abnormalities.^[11]

Several physiological mechanisms may explain the occurrence of non-dipping patterns. During normal

sleep, sympathetic activity decreases while parasympathetic activity increases, resulting in reduced blood pressure. Non-dipping may arise due to persistent nocturnal sympathetic activation, impaired autonomic regulation, endothelial dysfunction, altered sodium handling, psychological stress, poor sleep quality, or genetic predisposition.^[12]

One of the most significant findings of the present study was the higher AASI observed among non-dippers compared with dippers. This suggests that abnormal nocturnal blood pressure regulation is accompanied by increased arterial stiffness even before the onset of overt hypertension. AASI is considered an indirect measure of arterial compliance. Stiffer arteries exhibit reduced buffering capacity against pulsatile blood flow, resulting in altered relationships between systolic and diastolic blood pressure. Consequently, higher AASI values reflect diminished arterial elasticity and increased vascular stiffness.^[13]

The association between non-dipping status and elevated arterial stiffness observed in the present study is supported by previous research. Dolan et al. demonstrated that increased AASI predicts cardiovascular mortality independent of conventional risk factors.^[14] Li et al reported strong associations between AASI and target organ damage as well as cardiovascular risk.^[7]

Recent investigations have further highlighted the importance of nocturnal blood pressure patterns. Studies have shown that non-dipping status is associated with increased carotid intima-media thickness, left ventricular hypertrophy, microalbuminuria, and endothelial dysfunction.^[15,16] These abnormalities likely reflect cumulative vascular injury resulting from persistent nocturnal hemodynamic stress.

The absence of reverse dippers in the present study may be attributed to the relatively young age and healthy nature of the study population. Reverse dipping is more commonly observed in elderly individuals and in patients with hypertension, diabetes, chronic kidney disease, and established cardiovascular disease.^[17]

The observed relationship between increased AASI and non-dipping status may be bidirectional. Increased arterial stiffness can impair baroreceptor sensitivity and disrupt normal autonomic control mechanisms, thereby contributing to abnormal nocturnal blood pressure regulation. Conversely, sustained nocturnal elevation of blood pressure may promote vascular remodelling and accelerate arterial stiffening over time.^[18]

From a preventive cardiology perspective, the present findings are particularly relevant. Conventional office blood pressure measurements may fail to identify individuals with abnormal circadian blood pressure patterns. ABPM provides additional prognostic information by identifying non-dippers who may otherwise be considered healthy. Early recognition of such individuals may allow

implementation of lifestyle interventions aimed at reducing future cardiovascular risk.

Recent evidence emphasizes the role of sleep quality, stress management, regular physical activity, and dietary modifications in maintaining healthy circadian blood pressure rhythms. Young adults identified as non-dippers may particularly benefit from these preventive strategies.^[19]

An important strength of the present study was the use of 24-hour ambulatory blood pressure monitoring, which enabled assessment of both daytime and nocturnal blood pressure patterns rather than relying solely on clinic blood pressure measurements. The study also focused on a relatively young, healthy and normotensive population, allowing the relationship between nocturnal blood pressure regulation and arterial stiffness to be explored before the development of overt hypertension or clinically apparent cardiovascular disease. Furthermore, the use of AASI provided an objective, non-invasive estimate of arterial stiffness derived from routine ambulatory blood pressure recordings, thereby facilitating the identification of potential early vascular alterations in apparently healthy individuals.

The findings should, however, be interpreted in the context of certain limitations. The relatively small sample size may have limited the statistical power and generalizability of the findings. In addition, only male participants were included, and therefore the results cannot be directly extrapolated to females. The single-center setting may also limit the applicability of the findings to other populations with different demographic or environmental characteristics. The cross-sectional design allows identification of an association between non-dipping status and AASI but does not establish a temporal or causal relationship between the two variables. Moreover, arterial stiffness was assessed indirectly using AASI, without direct measurement by established vascular parameters such as carotid-femoral pulse wave velocity. Sleep quality and autonomic function, both of which may influence nocturnal blood pressure patterns, were also not specifically assessed in the present study.

Future studies involving larger and more diverse populations, including females and participants from multiple centres, would help determine the generalizability of these findings. Longitudinal studies incorporating direct measures of arterial stiffness, detailed assessment of sleep quality, and evaluation of autonomic function may further clarify the relationship between arterial stiffness and nocturnal blood pressure regulation and determine whether elevated AASI among non-dippers has long-term prognostic significance.

CONCLUSION

The present study demonstrated that approximately one-third of healthy normotensive young adult males

exhibited a non-dipping nocturnal blood pressure pattern. Non-dippers showed significantly higher Ambulatory Arterial Stiffness Index values compared with dippers, indicating increased arterial stiffness despite normal blood pressure levels. These findings suggest that abnormal nocturnal blood pressure regulation may represent an early marker of vascular dysfunction and future cardiovascular risk. Ambulatory blood pressure monitoring and AASI assessment may be useful tools for identifying young individuals at increased cardiovascular risk before the development of overt hypertension. Early detection of non-dipping status could facilitate timely preventive interventions aimed at reducing long-term cardiovascular morbidity.

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