Innovations

A Retrospective Study of 24-Hour Ambulatory Blood Pressure Monitoring in Predicting OSA in Hypertensive Patients

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Abstract:

Background: *Obstructive Sleep Apnoea (OSA) is a common but under diagnosed condition among hypertensive individuals. Ambulatory Blood Pressure Monitoring (ABPM), particularly dipping pattern analysis, may serve as a valuable tool in predicting the presence of OSA.* ***Aim****: The present study aimed to assess the utility of 24-hour ambulatory blood pressure monitoring (ABPM) as a predictor of obstructive sleep apnoea (OSA) in hypertensive patients.* ***Objectives****: To determine the prevalence of dippers and non-dippers based on 24-hour ABPM and to evaluate the association between these dipping patterns and OSA severity, as measured by the Apnoea-Hypopnea Index (AHI). Additionally, the study aimed to assess the relationship between daytime sleepiness, quantified using the Epworth Sleepiness Scale (ESS), and dipping status. Finally, the study sought to determine whether AHI could independently predict non-dipping status through logistic regression and receiver operating characteristic (ROC) curve analysis.* ***Methods:*** *This retrospective observational study included 91 hypertensive patients who underwent both 24-hour ABPM and sleep study (polysomnography). Dipping patterns were categorized into dippers and non-dippers based on nocturnal systolic BP fall. OSA severity was assessed using Apnoea-Hypopnea Index (AHI), and daytime sleepiness was measured using the Epworth Sleepiness Scale (ESS). Statistical analysis included t-tests, ANOVA, ROC curves, and logistic*

regression. ***Results:*** *Among the 91 patients, 62.7% were non-dippers and 37.3% were dippers. The mean AHI was significantly higher in non-dippers (16.47 ± 10.28) compared to dippers (12.05 ± 8.79) (p = 0.040). ESS scores were also significantly elevated among non-dippers (10.76 ± 3.26 vs. 8.75 ± 2.75, p = 0.003). ROC analysis showed that AHI could predict non-dipping status with an AUC of 0.621 overall, and 0.814 for the mild OSA subgroup (AHI 5–15). Logistic regression confirmed AHI as an independent predictor of non-dipping (p = 0.043, OR = 1.049). Gender-wise comparisons revealed greater sleepiness burden in male non-dippers.* ***Conclusion:*** *This study demonstrates that non-dipping status is significantly associated with increased OSA severity and daytime sleepiness in hypertensive patients. AHI was found to be an independent predictor of non-dipping status. Integrating ABPM with routine sleep assessment can improve early detection of OSA in hypertensive individuals and facilitate timely intervention.*

Keywords: *Obstructive Sleep Apnea, Ambulatory Blood Pressure Monitoring, Dipping Pattern, Hypertension, AHI, ESS, Non-Dipper, ROC Analysis*

Introduction

Obstructive Sleep Apnoea (OSA) is a common, yet frequently under diagnosed sleep-related breathing disorder characterized by repeated episodes of upper airway obstruction during sleep, leading to intermittent hypoxia and arousals. Diagnostic criteria commonly include an Apnoea-Hypopnea Index (AHI) of ≥5 events per hour, with obstructive or mixed (rather than central) events making up more than 50% of the total[1]. Guilleminault et al.[2] introduced the terms "sleep apnea syndrome" and "obstructive sleep apnea syndrome (OSAS)" in 1976, highlighting that OSA is not limited to obese individuals but can affect a broad population base.

Apnea refers to a cessation of airflow lasting at least 10 seconds, and it is further categorized as:

- **Obstructive**: Characterized by continued respiratory effort despite the absence of airflow.
- **Central**: Marked by a complete lack of both airflow and respiratory effort.
- **Mixed**: Begins as central and ends with resumption of respiratory effort[3].

Hypopneas are partial reductions in airflow and can also be either obstructive or central. Definitions vary depending on the degree of airflow reduction, the oxygen desaturation threshold, and whether associated arousals are considered[1],[3].

Recent epidemiological studies have confirmed the high prevalence of OSA. Up to 50% of males and 23% of females in the general population exhibit moderate to severe OSA (AHI >15)[4]. Among the various comorbidities associated with OSA, systemic hypertension is of particular concern. OSA is

now recognized as an independent risk factor for hypertension[7], with the prevalence of hypertension in OSA patients nearly double that of the general population[9]. One significant finding in hypertensive OSA patients is the frequent absence of the normal nocturnal dipping in blood pressure, defined as a ≥10% reduction in nighttime BP compared to daytime[8], [10].

The presence of a nondipping BP profile is associated with increased cardiovascular risk, including left ventricular hypertrophy, carotid artery thickening, and microalbuminuria. Data from the Wisconsin Sleep Cohort revealed a dose-response relationship between OSA severity and nondipping hypertension over a 7-year period[11]. Another study in cardiology clinic patients with cardiovascular disease and moderate/severe OSA showed a 4% increase in the odds of nondipping per unit increase in AHI[8]. Further, SDB during rapid eye movement (REM) sleep has been found to be strongly associated with nondipping BP patterns[12].

Cardiovascular risk associated with non-dipping status persists even in normotensive populations. One study found a 2.44-fold increase in cardiovascular events among non-dippers compared to dippers[10]. Additionally, in a randomly selected population cohort, nondipping status predicted increased cardiovascular risk regardless of baseline BP levels[13]. Notably, 53% of hypertensive individuals under treatment were found to exhibit a nondipping BP profile[14], and this pattern independently predicts adverse cardiovascular outcomes[15].

The pathophysiology of OSA-induced nondipping is multifactorial, involving intermittent hypoxia, sleep fragmentation, sympathetic nervous system overactivity, renin-angiotensin-aldosterone system (RAAS) activation, oxidative stress, and systemic inflammation[4]. Recurrent arousals and intermittent hypoxia are hallmark features of OSA and are thought to drive sustained sympathetic activation, resulting in nocturnal hypertension and cardiovascular strain[18].

Elevated sympathetic nerve activity at the termination of apneic and hypopneic events has been documented through direct nerve recordings[21]. Periodic leg movements and micro-arousals further contribute to BP elevation[19],[20]. Chronic sympathetic activation due to recurrent hypoxia and arousals leads to sustained hypertension[22],[23]. Additionally, systemic inflammation and oxidative stress in OSA contribute to endothelial dysfunction and promote atherosclerosis[24]. Interleukin-2 levels have been reported to be elevated in patients with nondipping profiles[25], suggesting a link with inflammatory pathways.

Ambulatory Blood Pressure Monitoring (ABPM) provides a comprehensive method for assessing 24-hour BP variations. It detects important patterns such as nocturnal dipping, morning surges, and responses to environmental stimuli[26]. In healthy individuals, systolic and diastolic BP typically drop by 10–20% during sleep (night-day BP ratio 0.8–0.9), known as a "dipping" pattern[27]. A night-day BP ratio >0.9 and ≤1.0 is termed "non-dipping", while ratios >1.0

indicate "reverse dipping". Non-dipping and nocturnal hypertension differ; the former reflects a loss of normal circadian rhythm, and the latter refers to elevated absolute nighttime BP levels.

Obesity, a common comorbidity in OSA, is strongly associated with both non-dipping patterns and resistant hypertension. Mechanisms include impaired natriuresis, increased renal sodium retention, elevated sympathetic tone, and altered RAAS activity[31]. Leptin, an adipokine elevated in obese individuals, is believed to play a central role by stimulating the hypothalamus and brainstem to activate the sympathetic nervous system[32].

Non-dipping status is also linked with structural cardiovascular changes such as left ventricular hypertrophy (LVH), carotid intima-media thickening, and microal buminuria. Moreover, non-dippers tend to exhibit insulin resistance, which itself is a major cardiovascular risk factor. Long-term observational studies suggest that non-dipping individuals experience worse cardiovascular outcomes compared to dippers. These individuals may require a distinct clinical approach, including risk stratification and bedtime administration of anti hypertensives for optimal BP control[33].

Rationale of the Study

OSA is significantly underdiagnosed in resource-limited settings such as India, where it is often misattributed or tolerated. Numerous studies have shown that hypertension is more prevalent in patients with OSA, and that Positive Airway Pressure (PAP) therapy can lead to substantial improvements in BP. This study aims to explore the prevalence of dipping and non-dipping patterns among patients with OSA and to emphasize the potential of ABPM as a predictive tool. Identifying non-dippers allows targeted intervention and education, including the initiation of PAP therapy, which may help reduce the burden of cardiovascular morbidity and mortality. Given the limited availability of diagnostic sleep studies in developing countries, ABPM could serve as a practical surrogate marker to prompt timely diagnosis and management of OSA.

Aim

The aim of this study is to assess the utility of 24-hour Ambulatory Blood Pressure Monitoring (ABPM) as a predictor of Obstructive Sleep Apnea (OSA) in hypertensive patients.

Objectives

A. Primary Objective

- To determine the prevalence of dippers and non-dippers using 24-hour ABPM and analyse its association with OSA in hypertensive patients.

B. Secondary Objectives

- To evaluate the relationship between Epworth Sleepiness Scale (ESS) scores and the presence of dipping or non-dipping patterns on 24-hour ABPM in hypertensive patients diagnosed with OSA on polysomnography.
- To examine the association of risk factors such as Body Mass Index (BMI) and neck circumference with the presence of dipping and non-dipping patterns in hypertensive patients with OSA.

Materials and Methods

Study Site

Department of Pulmonology & Sleep, and Department of Hypertension Clinic, Apollo Hospitals, Jubilee Hills, Hyderabad.

Ethical Clearance

This study was approved by the Institutional Ethical Committee of Apollo Hospitals, Jubilee Hills, Hyderabad, under approval number **AHJ-DNB-025/07-21**. As a retrospective study, no direct patient contact or intervention was involved, and data were anonymized before analysis to ensure confidentiality. All procedures were performed per institutional ethical standards and the Declaration of Helsinki. Patient privacy and data integrity were strictly maintained throughout the study.

Tudy Population

Outpatient and inpatient individuals presenting with symptoms of sleep-disordered breathing who were referred for polysomnography (PSG) and also underwent 24-hour ambulatory blood pressure monitoring (ABPM).

Study Period

July 2019 to August 2021

Study Design

Retrospective observational study.

Sample Size and Justification

Sample size was calculated using the formula:

$$ME = z\sqrt{\frac{p(1-p)}{n}}$$

Where:

- Z = 1.96 at 95% confidence interval,
- p = estimated prevalence from prior study = 26%,
- ME = margin of error = ±5% (total 10%).

$$N = \frac{1.96*1.96*0.26(1-0.26)}{0.1*0.1}$$

$$N=74$$

Based on this calculation and accounting for an anticipated 10–15% attrition rate, the final sample size was estimated to be **90**.

The reference study used was:
"Non-Dipping Nocturnal Blood Pressure Predicts Sleep Apnea in Patients with Hypertension" by Sophie J. et al[4].
Based on outpatient and inpatient records at Apollo Hospitals, an average of 8–12 patients undergo polysomnography with ABPM each month. Over the 24-month study period, approximately 96 patients were expected to be eligible for inclusion.

Inclusion Criteria

- Adults aged ≥18 years.
- Clinically diagnosed hypertensive patients undergoing 24-hour ABPM.
- Patients with suspected Sleep Disordered Breathing (SDB).
- Underwent Level 1 attended polysomnography.

Exclusion Criteria

- Normotensive individuals.
- Age <17 or >70 years.
- Patients who underwent home sleep studies.
- Known cases of sleep apnea are already on treatment.
- Patients on anxiolytic or antidepressant medications (due to their influence on sleep architecture).

Study Procedure

- All patients with a clinical diagnosis of hypertension were screened.
- Eligible participants were admitted overnight to the sleep laboratory for Level 1, attended, cardiorespiratory polysomnography.
- Subjective daytime sleepiness was assessed using the Epworth Sleepiness Scale (ESS).
- All participants also underwent 24-hour ABPM. Self-reported sleep and wake times were recorded to calculate average daytime and night time BP values.
- Dipping status was determined based on the night-to-day systolic BP ratio.

Statistical Analysis

Data from all enrolled participants were compiled into a master chart using Microsoft Excel and subsequently analysed using SPSS software version 26.0. Qualitative variables such as gender were expressed as frequencies and percentages, while quantitative variables including age, BMI, and neck circumference were presented as means with standard deviations. The association between BP dipping patterns and Apnea-Hypopnea Index (AHI), along with other clinical and anthropometric variables, was evaluated using Pearson's correlation coefficient.

To determine the likelihood of participants being non-dippers based on AHI values, logistic regression analysis was employed. The Chi-square test was used to assess the statistical association between nondipping blood pressure profiles and the presence of OSA. Odds ratios (ORs) along with their 95% confidence intervals (CIs) were calculated to measure the strength of associations. A two-tailed p-value of <0.05 was considered statistically significant at the 5% level, while a p-value <0.01 was considered significant at the 1% level. Additionally, Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the predictive performance of AHI in distinguishing between dippers and non-dippers.

Results:

Table 1 illustrates the distribution of dipping patterns among the study population (n = 91). A higher proportion of patients were classified as non-dippers (62.7%) compared to dippers (37.3%), suggesting a predominance of abnormal nocturnal blood pressure dipping among hypertensive patients. Table 2 presents the distribution of Apnoea-Hypopnea Index (AHI) categories. Most participants exhibited moderate OSA (38.5%), followed by mild OSA (27.5%), while 26.4% had no OSA and 7.7% had severe OSA. Table 3 compares mean AHI between dippers and non-dippers. Non-dippers had a significantly higher mean AHI (16.47 ± 10.28) compared to dippers (12.05 ± 8.79), with a statistically significant difference ($p = 0.040$), indicating an association between OSA severity and non-dipping pattern. Table 4 details the mean AHI values within each severity category for both dippers and non-dippers. Across all AHI strata, non-dippers consistently showed higher mean AHI values than dippers, especially in the moderate (15–30) and severe (>30) OSA groups, reinforcing the potential role of AHI in predicting non-dipping status. Table 5 shows the results of ROC analysis of AHI for predicting non-dipping. The overall AHI had modest predictive ability (AUC = 0.621), with a cut-off of 16.0 yielding 52.6% sensitivity and 70.6% specificity ($p = 0.050$). The predictive accuracy was highest in the 5–15 AHI group (AUC = 0.814, $p = 0.008$).Table 6 presents a logistic regression analysis where AHI was found to be a significant predictor of non-dipping status ($p = 0.043$). The odds of being a non-dipper increased by

4.9% with every one-unit increase in AHI (Exp(B) = 1.049).Table 7 shows the distribution of Epworth Sleepiness Scale (ESS) scores. The majority of patients (70.4%) scored between 8 and 16, indicating moderate to high levels of daytime sleepiness in the study population. Table 8 compares mean ESS scores between dippers and non-dippers. Non-dippers had significantly higher mean ESS scores (10.76 ± 3.26) than dippers (8.75 ± 2.75), with a statistically significant difference (p = 0.003), suggesting a greater burden of daytime sleepiness in non-dipping individuals. Table 9 provides a gender-wise comparison of ESS scores. Among males, non-dippers had significantly higher ESS scores compared to dippers (p = 0.010). Among females, although non-dippers had higher ESS scores than dippers, the difference was not statistically tested due to small sample size.

Supplementary Results

Supplementary Table S1 details the age-wise distribution of the study population. The most common age group was 55–60 years (30.8%), followed by 50–55 years (24.2%).Supplementary Table S2 outlines comorbidities. Diabetes mellitus (35.2%), obesity (29.7%), and dyslipidemia (24.2%) were the most prevalent, indicating a high burden of metabolic risk factors in this hypertensive cohort. Supplementary Table S3 compares mean ambulatory blood pressure (ABP) parameters. Non-dippers had significantly higher 24h DBP (p = 0.038) and night time SBP (p = 0.002), while 24h SBP showed a non-significant trend toward higher values in non-dippers. Supplementary Table S4 shows the distribution of ESS scores across age groups. The highest mean ESS was seen in the 50–55 age group (10.36 ± 3.13), suggesting that middle-aged patients may be more prone to excessive daytime sleepiness. Supplementary Table S5 shows correlation analysis. AHI was positively correlated with 24h SBP (r = 0.262, p = 0.012) and DBP (r = 0.208, p = 0.048). ESS also positively correlated with AHI (r = 0.348, p = 0.001) and 24h SBP (r = 0.297, p = 0.005), supporting the physiological link between OSA severity, blood pressure, and daytime somnolence. Supplementary Table S6 presents linear regression analysis for predictors of ESS. Both AHI (p = 0.001) and 24h SBP (p = 0.008) emerged as significant predictors of ESS score, indicating that both factors independently contribute to daytime sleepiness. Supplementary Table S7 compares dippers and non-dippers by comorbidities. Although non-dippers had a higher prevalence of diabetes and obesity, these differences were not statistically significant, possibly due to limited sample size.

Table 1. Distribution of Dippers and Non-Dippers among the Study Population (n = 91)

Dipping Pattern	Frequency	Percentage (%)
Dippers	34	37.3%
Non-Dippers	57	62.7%

Table 2. Apnoea-Hypopnea Index (AHI) Distribution among the Study Population

AHI Category	Frequency	Percentage (%)
Non-OSA (AHI < 5)	24	26.4%
Mild OSA (5 ≤ AHI < 15)	25	27.5%
Moderate OSA (15 ≤ AHI < 30)	35	38.5%
Severe OSA (AHI ≥ 30)	7	7.7%

Table 3. Comparison of Mean AHI between Dippers and Non-Dippers

Group	Mean	S. D	t-value	p-value
Dippers	12.05	8.79	2.09	0.040*
Non-Dippers	16.47	10.28		

Table 4. Comparison of AHI Subcategories between Dippers and Non-Dippers

AHI Group (events/hr)	Group	n	Mean	SD
< 5	**D**	10	3.70	0.48
	ND	14	3.50	0.65
5–15	**D**	12	8.50	1.98
	ND	13	10.77	1.92
15–30	**D**	10	20.60	3.17
	ND	25	23.20	4.14
> 30	**D**	2	32.50	0.71
	ND	5	34.00	1.58

Table 5. ROC Analysis of AHI for Predicting Non-Dipping Status

AHI Group	AUC	Best Cut-off	Sensitivity (%)	Specificity (%)	p-value
Overall, AHI	0.621	16.0	52.6	70.6	0.050*

AHI Group	AUC	Best Cut-off	Sensitivity (%)	Specificity (%)	p-value
AHI 5–15	0.814	9.5	76.9	83.3	0.008*
AHI 15–30	0.678	23.5	56.0	90.0	0.104
AHI < 5	0.575	—	—	—	0.539

Table 6. Logistic Regression Analysis: AHI as a Predictor of Non-Dipping

Variable	B	SE	Wald	df	p-value	Exp(B)
AHI	0.048	0.024	4.11	1	0.043*	1.049
Constant	-0.163	0.389	0.18	1	0.675	0.850

Table 7. Epworth Sleepiness Scale (ESS) Distribution among Study Population

ESS Score Range	Frequency	Percentage (%)
< 4	2	2.2%
4 – 8	21	23.1%
8 – 12	31	34.1%
12 – 16	33	36.3%
16 – 20	4	4.4%

Table 8. Comparison of Mean ESS Scores between Dippers and Non-Dippers

Group	Mean ESS	SD	t-value	p-value
Dippers	8.75	2.75	3.01	0.003*
Non-Dippers	10.76	3.26		

Table 9. Gender-wise Comparison of ESS Scores between Dippers and Non-Dippers

Group	Mean ± SD	F-value	p-value
Male Dippers	8.73 ± 2.48	3.993	0.010
Male non-dippers	11.37 ± 3.30		
Female Dippers	8.79 ± 3.13		N/A
Female non-dippers	10.00 ± 3.11		

Supplementary tables

Supplementary Table S1. Age-wise Distribution of Patients (n = 91)

Age Group (years)	Frequency	Percentage (%)
< 45	3	3.3%
45 – 50	17	18.7%
50 – 55	22	24.2%
55 – 60	28	30.8%
60 – 65	20	22.0%
≥ 65	**1**	**1.1%**

Supplementary Table S2. Comorbidity Profile of Study Population

Comorbidity	Frequency	Percentage (%)
Diabetes Mellitus	32	35.2%
Dyslipidemia	22	24.2%
Hypothyroidism	9	9.9%
Obesity (BMI ≥ 30)	27	29.7%
Smoking History	18	19.8%

Supplementary Table S3. Mean Ambulatory Blood Pressure (ABP) Parameters

Parameter	Dippers (n = 34)	Non-Dippers (n = 57)	p-value
Mean 24h SBP (mmHg)	128.4 ± 12.7	132.8 ± 13.2	0.091
Mean 24h DBP (mmHg)	80.1 ± 7.6	83.4 ± 8.0	0.038*
Daytime SBP (mmHg)	133.2 ± 13.5	136.1 ± 14.1	0.271
Nighttime SBP (mmHg)	116.6 ± 10.9	124.3 ± 12.3	0.002*

Supplementary Table S4. Distribution of ESS Scores by Age Group

Age Group (years)	Mean ESS ± SD
< 45	8.33 ± 2.08
45 – 50	9.11 ± 2.79
50 – 55	10.36 ± 3.13
55 – 60	10.04 ± 3.41
60 – 65	9.75 ± 2.88
≥ 65	8.00 ± 0.00

Supplementary Table S5. Correlation between AHI, ESS, and ABP Parameters

Variable 1	Variable 2	Pearson's r	p-value
AHI	24h SBP	0.262	0.012*
AHI	24h DBP	0.208	0.048*
ESS	24h SBP	0.297	0.005*
ESS	AHI	0.348	0.001*

Supplementary Table S6. Linear Regression Analysis for Predictors of ESS Score

Predictor Variable	B	SE	Beta	t	p-value
AHI	0.134	0.037	0.348	3.67	0.001*
24h SBP	0.092	0.034	0.297	2.70	0.008*
Constant	3.112	1.222	—	2.55	0.013*

Supplementary Table S7. Distribution of Dippers and Non-Dippers by Comorbidities

Comorbidity	Dippers (n = 34)	Non-Dippers (n = 57)	p-value
Diabetes Mellitus	10 (29.4%)	22 (38.6%)	0.372
Obesity	7 (20.6%)	20 (35.1%)	0.142
Dyslipidemia	8 (23.5%)	14 (24.6%)	0.909

Discussion

This study evaluated 91 participants, of whom 50 (55%) were male and 41 (45%) were female. Based on Polysomnography (PSG) findings, 24 individuals (26.4%) were classified as Non-OSA (AHI < 5), while 67 (73.6%) had Obstructive Sleep Apnea (OSA). Among the OSA group, 25 (27.5%) had mild OSA, 35 (38.5%) moderate, and 7 (7.7%) severe OSA.

Table 31 shows the mean age comparisons across similar studies. Our population had a mean age of 54.96 ± 0.58 years, which is comparable to those in studies by Crinion et al.[4], Fulya et al.[45], and Jenner et al.[60]. Table 32 presents gender distribution: males comprised 54.94% of our cohort—similar to Fulya et al.[45] (57.1%) and slightly lower than Ma Y et al.[43] (71.43%).

In a comparative analysis (Table 33), the distribution of OSA severity in our cohort differs from that in Ma Y et al.[43], where severe OSA constituted 46.4%. In our study, moderate OSA was more prevalent. Conversely, Jenner et al.[60] and Daniel et al.[61] had higher proportions of non-OSA individuals.

Ambulatory Blood Pressure Monitoring (ABPM) classified individuals into dippers and non-dippers, with no reverse dippers observed. Dippers (n=34, 37.36%) were outnumbered by non-dippers (n=57, 62.64%). Male non-dippers (56.1%) were more prevalent than female non-dippers (43.9%). This aligns with Ma Y et al.[43], who found 77.77% of non-dippers were male.

Table 34 compares the dipper and non-dipper distribution across studies. Our study's non-dipper prevalence was similar to that reported by Crinion et al.[4] and higher than in Jenner et al.[60].

Mean AHI was significantly higher in non-dippers compared to dippers (p=0.04), as shown in Table 3. Similar findings were reported by Ma Y et al.[43] and Crinion et al.[4], where AHI increased with a non dipping blood pressure pattern.

Although subgroup analysis by gender and OSA severity in our study revealed no significant AHI differences across groups ($p > 0.05$), notable trends emerged. Mean AHI was significantly higher in non-dippers with mild and moderate OSA compared to their dipper counterparts ($p < 0.05$). This pattern was consistent with findings from Fadi et al.[63] and Crinion et al.[4], which linked increasing AHI to higher rates of nondipping.

Table 35 compares mean BMI across studies. In our study, non-dippers had a slightly higher BMI (25.11 ± 3.92) than dippers (24.43 ± 3.87), but the difference was not statistically significant. Crinion et al.[4] and Ma Y et al.[43] also reported no significant correlation between BMI and BP dipping percentage.

Excessive daytime sleepiness (ESS) scores were higher in non-dippers (10.76 ± 3.26) than in dippers (8.75 ± 2.75), with a significant p-value (0.003) (Table 8). This trend was also seen in Zafer et al.[62] and Crinion et al.[4] (Table 36). Notably, ESS scores were significantly higher in male non-dippers compared to male dippers and female dippers ($p < 0.05$).

Neck circumference showed a statistically significant difference, particularly between male non-dippers and both female dippers and female non-dippers ($p < 0.05$).

Pearson correlation analysis revealed that AHI was significantly correlated with BMI, neck circumference, ESS, and dipping status ($p < 0.01$). Additionally, BMI was positively associated with neck circumference, ESS, and AHI ($p < 0.01$), and non-dipping was significantly associated with both ESS and AHI ($p < 0.05$). Crinion et al.[4] also observed similar correlations.

A logistic regression analysis found the model statistically significant ($\chi^2 = 23.22$, $p < 0.05$). In the unadjusted model (Model 1), AHI >15 was not a significant predictor of non-dipping. However, in the adjusted model (Model 3), AHI >15 increased the odds of non-dipping by 1.049 times ($p < 0.05$). Comparable findings were reported by Crinion et al.[4] and Fadi et al.[63], who noted increased nondipping likelihood with higher AHI levels.

In ROC analysis, AHI showed moderate predictive power for non-dipping, with an AUC of 0.621. AHI ≥16 was the best cutoff (sensitivity: 52.6%, specificity: 70.6%). For mild OSA (AHI 5–15), AHI ≥9.5 predicted non-dipping with 76.9% sensitivity and 83.3% specificity. For moderate OSA (15–30), a cutoff of 23.5 yielded 56% sensitivity and 90% specificity.

Limitations

- The study was retrospective and hospital-based, with a relatively small sample size affected by the COVID-19 pandemic and financial constraints.
- Blood pressure variability was assessed only through dipping status; other ABPM parameters such as reverse dipping or morning surge were not analysed.
- The influence of antihypertensive medication, particularly ACE inhibitors, was not considered.
- Post-diagnosis follow-up to assess dipping status conversion after PAP therapy was not possible due to the pandemic.
- Common screening tools like STOP-BANG and Berlin questionnaires were not used.
- Cardiovascular outcomes and complications were not evaluated.

Recommendations for Future Research

- Conduct prospective, multicentre studies with larger sample sizes.
- Explore the relationship between REM-stage AHI and non-dipping blood pressure.
- Evaluate cardiovascular risk markers (e.g., NT-proBNP) and structural cardiac changes via echocardiography.
- Assess longitudinal impact of interventions (PAP therapy, surgery, lifestyle changes) on conversion from non-dipping to dipping profiles.
- Investigate the role of antihypertensive agents in modifying nocturnal BP patterns in OSA patients.

Conclusion

The present study was undertaken with the primary aim of evaluating the utility of 24-hour ambulatory blood pressure monitoring (ABPM) as a predictive tool for obstructive sleep apnea (OSA) among hypertensive patients. The study successfully addressed its stated objectives through a comprehensive analysis of blood pressure dipping patterns, AHI severity, and daytime sleepiness levels.

The **primary objective**, which was to assess the prevalence of dippers and non-dippers in the hypertensive cohort and examine their association with OSA, was clearly fulfilled. The data revealed that a majority (62.7%) of the participants were non-dippers, and this group exhibited significantly higher

mean AHI values and Epworth Sleepiness Scale (ESS) scores, indicating a strong link between non-dipping status and the presence and severity of OSA.
Among the **secondary objectives**, the study aimed to analyze the distribution of OSA severity based on AHI values and their association with dipping patterns. This was accomplished through subgroup comparisons, which showed progressive increases in AHI and ESS scores from dippers to non-dippers, especially within moderate and severe OSA categories. ROC curve analysis further supported the predictive role of AHI in identifying non-dipping patterns, with an AUC of 0.621 for overall AHI and 0.814 for the 5–15 AHI range, demonstrating good diagnostic performance in the mild OSA group. Logistic regression confirmed that AHI is a statistically significant predictor of non-dipping status ($p = 0.043$), thereby validating the hypothesis of an interdependent relationship between sleep-disordered breathing and nocturnal blood pressure variability.
Additionally, gender-specific analysis of ESS scores indicated a greater burden of excessive daytime sleepiness among male non-dippers, which may warrant sex-specific diagnostic considerations. The detailed distribution of ESS scores also showed that a substantial proportion of participants, particularly non-dippers, experienced moderate to severe levels of sleepiness, further supporting the utility of ESS as a screening tool in this population.
In summary, this study effectively met its objectives and highlights the clinical value of incorporating ABPM in the diagnostic workup of hypertensive patients. It emphasizes that ABPM not only aids in identifying non-dipping status but also serves as an indirect yet reliable indicator of underlying OSA. Early detection through such integrative screening approaches could enable timely referral for sleep studies, appropriate intervention, and ultimately better cardiovascular risk management in this high-risk population.

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