

Non-dipping hypertension as a clue to undiagnosed obstructive sleep apnea: A case report

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Abstract

Obstructive Sleep Apnea (OSA) is a common but frequently underdiagnosed disorder and an important secondary cause of hypertension. A non-dipping nocturnal blood pressure pattern detected by Ambulatory Blood Pressure Monitoring (ABPM) has been associated with increased cardiovascular risk and may provide a clinical clue to underlying OSA. We report the case of a 74-year-old female with long-standing, poorly controlled hypertension and multiple cardiovascular comorbidities evaluated in a family medicine outpatient setting. Twenty-four-hour ABPM revealed a paradoxical nocturnal rise in blood pressure with marked systolic variability, consistent with a non-dipping pattern. Clinical assessment identified obesity, increased neck circumference, loud snoring, daytime sleepiness, and a high STOP-BANG score. Polysomnography subsequently confirmed severe obstructive sleep apnea. Continuous Positive Airway Pressure (CPAP) therapy led to a marked reduction in apnea-hypopnea index and improvement in nocturnal respiratory parameters. During long-term follow-up, combined CPAP therapy, lifestyle modification, optimization of antihypertensive treatment, and management of additional secondary causes of hypertension resulted in partial normalization of nocturnal blood pressure patterns and improved blood pressure control. This case underscores the clinical value of ABPM in prompting further evaluation for OSA in patients with atypical or uncontrolled hypertension.

Keywords: Obstructive sleep apnea; Non-dipping hypertension; Ambulatory blood pressure monitoring; Secondary hypertension; Continuous positive airway pressure.

Introduction

Obstructive Sleep Apnea (OSA) is a respiratory disorder characterized by recurrent partial or complete upper airway obstruction during sleep, resulting in apnea ($\geq 90\%$ airflow reduction for ≥ 10 seconds) or hypopnea ($\geq 30\%$ airflow reduction for ≥ 10 seconds with desaturation or arousal). According to The AASM Manual for the Scoring of Sleep and Associated Events, respiratory events are scored when associated with a $\geq 3\%$ oxygen desaturation and/or arousal [1]. These events lead to impaired gas exchange,

intrathoracic pressure changes, and sleep fragmentation. OSA is an independent risk factor for several cardiovascular conditions, including hypertension, stroke, coronary artery disease, heart failure, and chronic kidney disease, and represents the most common secondary cause of resistant hypertension [2,3].

Blood Pressure (BP) follows a circadian rhythm with a normal nocturnal decline of 10–20% (dipping), whereas a reduction of less than 10% defines a non-dipping pattern, which is associated with increased cardiovascular risk [4]. Twenty-four-hour Ambulatory Blood Pressure Monitoring (ABPM) is essential for assessing these circadian BP abnormalities, particularly in patients with suspected OSA [5].

This report presents a case in which a non-dipping BP pattern on ABPM prompted the diagnosis of OSA in a 74-year-old female with complex cardiovascular comorbidity.

Case Report

A 74-year-old female presented in October 2019 with uncontrolled hypertension in family medicine outpatient clinic. Her medical history included hypertension (Grade I), hypertensive retinopathy (Grade I), dyslipidemia, nodular goiter, generalized atherosclerosis, severe peripheral arterial disease, bilateral occlusion of the superficial femoral arteries, carotid atherosclerosis, vertebrobasilar insufficiency, cervical spondylosis, and intervertebral disc pathologies (C3/4, C4/5, C6/7). She quit smoking in 2002 after 15 years of 20 cigarettes/day and denied alcohol consumption. Upon examination, her Epworth Sleepiness Scale score was 10. The STOP-BANG score is a screening tool used to assess the risk of Obstructive Sleep Apnea (OSA). It consists of 8 yes/no questions - each “Yes” scores 1 point, for a total score between 0 and 8. The STOP-BANG score in this case was calculated to be 5 points, indicating a high risk of OSA.

The patient’s BMI (34.1 kg/m² at the initial visit and 30.5 kg/m² at the last visit) and neck circumference (40 cm at the initial visit and 39 cm at the last visit) (Table 1).

The patient reported loud snoring and occasionally choking during sleep. At the initial 24-hour Ambulatory Blood Pressure Monitoring (ABPM) performed in the family medicine outpatient clinic, paradoxical nocturnal blood pressure elevation and significant systolic blood pressure variability were observed. Subsequently, a split-night polysomnography was conducted at Sleep Center on December 11, 2019, to assess the severity of sleep-disordered breathing and the effectiveness of Continuous Positive Airway Pressure (CPAP) therapy.

The baseline apnea–hypopnea index (AHI) was 30.8 events per hour, the Oxygen Desaturation Index (ODI) 48.3 events per hour, and the Arousal Index (AI) 19.2 events per hour, consistent with severe obstructive sleep apnea. Following the initiation of CPAP therapy, AHI, ODI, and AI were markedly reduced to 1.4, 3.1, and 5.3 events per hour, respectively, demonstrating substantial improvement in nocturnal respiratory function. The patient used CPAP regularly (average of 5.7 hours/night, >70% of nights). No significant side effects were reported. These findings indicate a substantial improvement in nocturnal respiratory parameters following CPAP therapy in this patient. The patient experienced a 10 kg weight reduction during follow-up, which may have contributed to the observed improvements alongside CPAP therapy (Table 1).

Table 1: Longitudinal changes in patient demographic and health parameters.

Parameter	Initial Visit (Oct 17, 2019)	Last Visit (Mar 21, 2024)	Change
Age (years)	69	74	+5
Body weight (kg)	94	84	-10
Height (m)	1.66	1.66	0
BMI (kg/m ²)	34.11	30.5	-3.61
Waist circumference (cm)	108	110	+2
Hip circumference (cm)	112	112	0
Arm circumference (cm)	36	35	-1
Neck circumference (cm)	40	39	-1
Heart rate (bpm)	63	65	+2
SBP (mean, mmHg)	141	137	-4
DBP (mean, mmHg)	79	76	-3
SBP variability	20.0	13.0	-7.0
DBP variability	10.8	7.7	-3.1
cSBP (mmHg)	167	142	-25
cDBP (mmHg)	85	76	-9
Aix@75 (%)	31	32	+1
Peripheral resistance (s*mmHg/ mL)	1.5	1.3	-0.2
PWV (m/s)	10.5	11	+0.5
Epworth Sleepiness Scale	10	9	-1

Further diagnostics in 2022 identified bilateral renal artery stenosis, managed via stent placement. Subsequent ABPM showed partial blood pressure regulation. Over the observation period, multiple clinical and hemodynamic parameters improved, whereas some remained unchanged (Table 1).

Over the course of five years, antihypertensive therapy has been adjusted multiple times. Latest prescribed therapy: bisoprolol 5 mg (0+1+0), moxonidine 0.4 mg (0+1+0), lercanidipine 10 mg (1+0+1), spironolactone 25 mg (1+0+0), furosemide 40 mg (1+0+0 every 3rd day) with KCl 500 mg.

Over time, improvements were observed in peripheral and central blood pressure levels, blood pressure variability, and nocturnal blood pressure patterns (Table 1, Figure 1).

ABPM was performed using a clinically validated Mobil-O-Graph PWA device with Hypertension Management Software CS (ANSI/AAMI/ISO 81060-2:2018).

Evolution of nocturnal systolic and diastolic blood pressure dipping patterns over time. Initially classified as a reverse dipper in October 2019, the patient transitioned to a non-dipping pattern following CPAP initiation and antihypertensive therapy adjustment, with partial normalization observed in 2022 and mild regression by 2024.

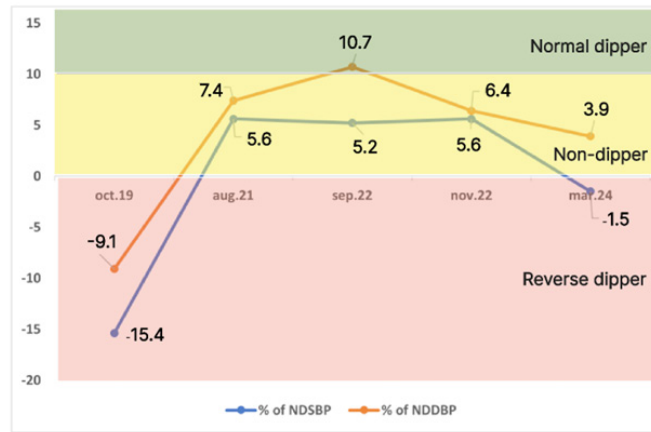


Figure 1: Nocturnal dipping pattern of systolic and diastolic BP over time in a patient with OSA.

Discussion

OSA affects about 20% of adults, with 6–7% experiencing severe forms. Prevalence increases with age, obesity (40–60% in obese men), and male sex. However, it is also seen in women, lean individuals, and the elderly [6].

Obesity remains the key modifiable risk factor for Obstructive Sleep Apnea (OSA), with a clear link between increased Body Mass Index (BMI) and OSA severity. Even modest weight gain significantly raises the Apnea-Hypopnea Index (AHI), while weight reduction lowers the risk of developing OSA. Central obesity markers, particularly waist and neck circumference, show even stronger associations. Neck circumference over 17 inches (43.18 cm) in men and 16 inches (40.64 cm) in women is considered a threshold for increased risk and may serve as a better predictor of OSA severity than BMI, showing strong correlations with AHI and oxygen desaturation [7].

As observed in the reported case, OSA may manifest with nocturnal symptoms such as snoring, gasping, and fragmented sleep, as well as daytime symptoms including excessive sleepiness, fatigue, and mood changes. OSA is an independent risk factor for multiple cardiovascular diseases, largely due to mechanisms such as intermittent hypoxia, increased sympathetic tone, and inflammation. Chronic OSA may lead to systemic issues, including cardiovascular and metabolic dysfunctions [8,9].

OSA is linked to increased sympathetic activity and hypertension (50% prevalence in OSA patients), contributing to resistant hypertension and endothelial dysfunction [10]. Non-dipping blood pressure patterns are common in OSA, significantly increasing cardiovascular risk [11].

Diagnosis relies on clinical history, screening tools (Epworth Sleepiness Scale, STOP BANG), and polysomnography - the gold standard - using the Apnea-Hypopnea Index (AHI) to assess severity of OSA [12]. The combination of ABPM findings and polysomnography may facilitate earlier identification of OSA in patients with hypertension, allowing for timely intervention. This approach is particularly beneficial given the underdiagnosis of OSA, with up to 90% of cases remaining undetected [13].

CPAP therapy has been shown to improve several physiological parameters and reduce blood pressure and arrhythmias; however, its impact on major cardiovascular outcomes may depend on adherence, symptom burden, and comorbid conditions [14].

In reported case, comprehensive lifestyle modifications, optimized antihypertensive therapy, renal artery stenting, and CPAP implementation collectively improved blood pressure control, nocturnal dipping, and OSA-related symptoms, consistent with findings from prior studies [15].

Conclusion

Abnormal nocturnal blood pressure patterns detected by ABPM, including non-dipping and reverse dipping profiles, may represent an important early indicator of obstructive sleep apnea in patients with hypertension. Recognition of these patterns can facilitate timely referral for sleep evaluation and initiation of appropriate therapy. This case underscores the role of ABPM as a valuable screening adjunct in the diagnostic pathway of OSA and highlights the consequences of delayed diagnosis on cardiovascular target organ damage.

Abbreviations: ABPM: Ambulatory Blood Pressure Monitoring; AHI: Apnea–Hypopnea Index; AI: Arousal Index; Aix@75: Augmentation Index normalized to 75 beats per minute; BMI: Body Mass Index; BP: Blood Pressure; cDBP: Central Diastolic Blood Pressure; CPAP: Continuous Positive Airway Pressure; cSBP: Central Systolic Blood Pressure; DBP: Diastolic Blood Pressure; ESS: Epworth Sleepiness Scale; KCl: Potassium Chloride; ODI: Oxygen Desaturation Index; OSA: Obstructive Sleep Apnea; PSG: Polysomnography; PWV: Pulse Wave Velocity; SBP: Systolic Blood Pressure; STOP-BANG: Snoring, Tiredness, Observed apnea, high blood Pressure, Body mass index, Age, Neck circumference, Gender.

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Ethics approval and consent to participate: This study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Public Institution Health Centre of Canton Sarajevo. Written informed consent was obtained from the patient for participation in the study and publishing a case report.

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