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ORIGINAL ARTICLE

Diastolic Pressure Dipping as a Predictor of Cardiovascular Complications in Patients with Obstructive Sleep Apnea

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ABSTRACT

Background: Patients with obstructive sleep apnea have a higher prevalence of nocturnal blood pressure nondippers. This lack of fluctuation in circadian blood pressure is a significant risk for end-organ damage from hypertension. So, for assessment of cardiovascular problems in obstructive sleep apnea, diastolic blood pressure nondipping measured by Ambulatory Blood Pressure Monitoring (ABPM) is helpful in addition to Holter electrocardiogram (ECG) and echocardiography. So we aimed to evaluate the predictive value of diastolic blood pressure; measured by ABPM, for cardiovascular complications in patients with Obstructive sleep apnea (OSA). **Methods:** This study included 64 patients diagnosed with OSA. All the patients were subjected to bedside trans-thoracic echocardiology, ambulatory blood pressure monitoring, resting ECG and Holter 24-hour ECG. **Results:** There was a positive association between the presence of Diabetes mellitus (DM), hypertension, Body mass index (BMI) and grades of OSA. Oxygen saturation (SpO2) was lower in patients with severe OSA than in mild and moderate cases. ABPM 24h detected masked hypertension in approximately 45% of included patients. There was a positive correlation between night-time average diastolic Blood pressure (BP) and BMI, interventricular hypertension, The apnea-hypopnea index (AHI), abnormal septum thickness (IVST), and diastolic dysfunction. Regarding overall detected cardio-vascular complications, diastolic dysfunction was detected in all patients. 65.6% had abnormal IVST, 40.625% had mild tricuspid regurgitation, 18.75% had ischemia and 32.8% had Atrial fibrillation (AF). All patients with regional wall motion abnormality had severe OSA. 91.7% of patients with Ischemic changes had severe OSA. **Conclusions:** ABPM 24h is considered a valuable tool in the detection of masked hypertension in non-dipper OSA patients. OSA is associated with a higher incidence of Cardiovascular disease (CVD) that increases with increasing degree of OSA.

Keywords: Diastolic pressure; Complication; Obstructive sleep apnea.

INTRODUCTION

With over 17 million fatalities yearly, cardiovascular illnesses collectively are the world's largest cause of death. These illnesses include vascular disorders of the brain, illnesses of the heart, and illnesses of the vessels [1].

The condition known as obstructive sleep apnea (OSA) is characterized by periodic blockage of the upper airway during sleep. This blockage can cause partial or complete cessation of airflow, which in turn leads to increased negative intrathoracic pressure, repeated ventilation attempts, intermittent hypoxia, and fragmentation of sleep [2].

Excessive relaxation of the throat muscles, followed by inspiratory efforts against the closed glottis, is linked to apneas. This ultimately causes the patient to become aroused, which is linked to sympathetic activation and hyperventilation. By lowering the arterial partial pressure of carbon dioxide (CO2) approaching the apnea threshold, hyperventilation in turn causes the subsequent apnea. Arterial oxygen desaturation is important in this situation because it reduces the difference between the eupneic and apneic CO2 pressure levels. This makes the CO2-controlled breathing regulation more unstable and therefore more prone to periodic breathing [3].

One prominent cardiovascular risk factor is OSA. Cardiovascular disorders are more common in people with OSA and are linked to lower functional outcomes as well as higher mortality rates [4].

While the patient sleeps, the blood pressure of the patient typically decreases by 10% to 15% from its daily reading. One term for this diurnal decrease in blood pressure (BP) is "dipping." Nonetheless, some people are referred to as non-dippers since they do not experience a nocturnal decrease in blood pressure. It has been observed that individuals with obstructive sleep apnea [5] and essential hypertension [6] had higher nondipper prevalence. There are theories that the nightly decline in blood pressure is a restorative physiological mechanism [7] and that one major risk factor for hypertensive end-organ damage may be the absence of circadian blood pressure fluctuation [8].

OSA frequently causes systemic hypertension, which has a significant impact on cardiovascular health. Additionally, there may be an increased risk of cardiovascular disease incidence and faster development with altered blood pressure (BP) variability [9].

METHODS

In this cohort study, 64 patients who had already received an OSA diagnosis were chosen from the cardiology, chest, and sleep lab departments of the Zagazig University Hospitals in Egypt.

Diagnosis of the OSA was already done at the sleep lab at the pulmonology department. OSA is often suspected based on symptoms and confirmed with diagnostic testing. Diagnostic testing can be performed by overnight in-laboratory, multichannel polysomnography, or home sleep apnea tests. Diagnosis requires the patient to have (1) reported nocturnal breathing disturbances (snoring, snorting, gasping, or breathing pauses during sleep) or symptoms of daytime sleepiness or fatigue occurring despite sufficient opportunity to sleep and unexplained by other medical conditions and (2) an Apnea Hypopnea Index (AHI) or Respiratory Event Index ≥5. OSA may be

diagnosed in the absence of symptoms if the Apnea Hypopnea Index (AHI) or Respiratory Event Index is ≥15 episodes per hour. Empirical categorization is based on the Apnea Hypopnea Index (AHI) or Respiratory Event Index of 5 to <15 (mild), 15 to 30 (moderate), and >30 (severe). However, a singular focus on the event frequency (Apnea Hypopnea Index (AHI)/Respiratory Event Index) does not capture other important aspects of OSA pathophysiology such as the degree of hypoxemia, event duration, temporal distribution of events across the sleep cycle, extent of sleep fragmentation, and presence of excessive daytime sleepiness. Recent research identified hypoxia burden as a predictor for increased cardiovascular disease (CVD) risk with other polysomnography-derived measures (e.g., loop gain, neuromuscular collapsibility) useful for identifying subgroups who may respond differently to alternative OSA treatments. Wearable technologies are progressively being adopted as diagnostic tools but need more validation [10].

Pregnant woman, <18 or >95 years of age, within the last six months, the patient experienced signs of an acute stroke or myocardial infarction, the diseases or factors causing secondary hypertension were evident in the patients, ABPM was intolerable to the patients, the patient's medical histories included substantial systemic disease, congestive heart failure, liver failure, kidney failure, and any kind of arrhythmia, unable to participate in polysomnography, existence of serious mental illnesses, coexistence of inadequate breathing and heart valve problems were excluded from the study.

Informed written consent was obtained from all participants. The study was approved by the ethical committee of the Faculty of Medicine, Zagazig University (IRB number 9417).

Following the acquisition of informed consent, every patient endured the following: A complete medical history, a clinical examination that includes vital signs and a local cardiac examination, as well as laboratory testing that includes arterial blood gases (ABG), lipid profiles (cholesterol, triglyceride (TG), low-density lipoproteins (LDL) & High-density lipoprotein (HDL), hemoglobin & Total Leucocyte Count (TLC), serum Potassium (K+) & Calcium ions (Ca2+), D-Dimer, erythrocyte sedimentation rate (ESR), and C-reactive protein(CRP).

Obstructive sleep apnea (OSA) is diagnosed based on clinical symptoms discovered during a physical examination and medical history. Patients who exhibit clinical symptoms and a physical examination and are suspected of having OSA should have sleep testing done. For the diagnosis of OSA, overnight polysomnography is the gold standard of sleep testing.

All research participants had bedside trans-thoracicechocardiology (TTE), ambulatory blood pressure monitoring, resting ECG, and Holter 24-hour ECG. A follow-up using an echocardiogram, resting ECG, and Holter ECG were performed for three months.

Statistical analysis:

The SPSS software arranged the data. Two-tailed tests were used to determine the significance of parametric variables, which are defined as means plus two standard deviations. The chi-square test was used to determine the significance of the non-

parametric variable after its frequencies has been assessed.

RESULTS

This study the included 64 non-dipper patients with OSA. The included patients' average age was 46.5 years, and their average body mass index was 37.65. 43 of included patients (67.2%) were female. 50% of included patients had grade 2 obesity. 42.2% had moderate OSA, 32.8% had severe OSA and 25.0% had mild OSA. There was a positive association between socio-demographic parameters and grades of OSA. Weight and BMI were greater in severe OSA patients compared to mild and moderate OSA patients. All patients with grade 3 obesity had severe OSA, while no patients with grade 1 obesity had severe OSA (Table 1).

Resting systolic/diastolic BP was greater in those with severe OSA than in those with mild to moderate instances. Compared to mild and moderate OSA patients, those with severe OSA had greater resting heart rate (HR) and Respiratory rate (RR) cases. SPO2 on Rheumatoid arthritis (RA) was lower in patients with severe OSA than in mild and moderate cases. Regarding Transthoracic Echocardiography, Posterior wall thickness (PWT), left atrium (LA) and pulmonary venous systolic pressure (PVSP) were higher compared to individuals with mild or moderate OSA, in patients with severe OSA. Compared to patients with mild or moderate OSA, those with severe OSA had decreased Tricuspid Annular Plane Systolic Excursion (TAPSE) (Table 2).

There was a significant increase in cases of hypertension detected using ABPM 24h compared to cases detected during clinical examination. The total systolic and diastolic blood pressure increased statistically as compared to Resting systolic/diastolic BP. ABPM 24h detected masked hypertension in approximately 45% of included patients (table 3).

There was a positive correlation between night-time average diastolic BP and abnormalIVST, and diastolic Dysfunction as in table (4).

There was a significant increase in cardiovascular complications detected by Holter ECG than resting ECG (figure 1).

Regarding overall detected cardio-vascular complications, diastolic dysfunction was detected in all patients. 65.6% had abnormal IVST, 40.625% had mild tricuspid regurgitation, 18.75% had ischemia and 32.8% had AF. Regarding grades of OSA, 80.8% of patients with mild tricuspid regurgitation and 75% of patients with mild mitral regurgitation had severe OSA. All patients with regional wall motion abnormality had severe OSA. 91.7% of patients with Ischemic changes had severe OSA (table 5).

Table 1: Baseline data among studied patients

	Overall (N=64)	Mild OSA (N=16)	moderate OSA(N=27)	severe OSA (N=21)	P value
Age	46.56±12.38 years	48.19±11.64 years	44.52±11.39 years	47.95±14.25 years	.536
Weight	105.16±11.60 Kg	98.50±6.23 Kg	101.93±9.18 Kg	114.38±12.11*# Kg	.000
Height	167.58±7.99 Cm	172.44±4.69 Cm	166.85±7.70 Cm	164.81±8.94*# Cm	.011
BMI	37.65±4.01 Kg/m^2	33.12±1.32 Kg/m^2	36.87±1.39* Kg/m^2	42.09±3.00*# Kg/m^2	.000
Sex					
Female	43 (67.2%)	7(16.30%)	21(48.80%)	15(34.90%)	0.063
Male	21 (32.8%)	9(42.90%)	6(28.60%)	6(28.60%)	
Obesity					
Grade 1	16 (25 %)	14(87.50%)	2(12.50%)	0(0.00%)	.000
Grade 2	32 (50 %)	2(6.30%)	25(78.10%)	5(15.60%)	
Grade 3	16 (25 %)	0(0.00%)	0(0.00%)	16(100.00%)	
comorbidities					
Diabetes mellitus	48 (75 %)	3 (6.30%)	25(52.10%)	20 (41.70%)	0.00
Hypertension	28 (43.8 %)	0 (0.0%)	7(25.0%)	21(75.0%)	0.00
Dyslipidemia	64 (100 %)	16 (100 %)	27 (100 %)	21 (100 %)	…..
Stroke	1 (1.6 %)	1 (100.0%)	0(0.0%)	0(0.0%)	.250
TIA	11 (17.2 %)	2(18.2%)	3(27.3%)	6(54.5%)	.304

* indicates a statistically significant difference with mild OSA, # indicates a statistically significant difference with moderate OSA, one-way ANOVA followed by Duncan test,
BMI: body mass index. OSA: obstructive sleep apnea. TIA: transient ischemic attack.

Table 2: Clinical Examinations and Parameters of Echocardiography

Clinical Examination	Overall (N=64)	Mild OSA (N=16)	moderate OSA(N=27)	severe OSA (N=21)	P value
Resting systolic BP	141.1±18.63 mmHg	128.8±5.0 mmHg	130±10.6 mmHg	164.8±7.3*# mmHg	.000
Resting diastolic BP	87.58±12.018 mmHg	81.3±5.0 mmHg	80.6±6.8 mmHg	101.4±8.7*# mmHg	.000
Resting HR	95.64±8.126 bpm	84.5±2.7 bpm	95.5±3.0* bpm	104.3±3.5*# bpm	.000
Temperature	36.80±.118 C	36.8±0.1 C	36.8±0.1 C	36.8±0.1 C	.434
RR	19.35±3.9 Breath/min	14.7±0.8 Breath/min	18.3±1.1* Breath/min	24.2±1.5*# Breath/min	.000
SPO2 on RA	94.82±2.16	97.5±0.7	95.2±0.8*	92.3±0.8*#	.000
NormalHeart sounds	64 (100%)	16 (100%)	27 (100%)	21 (100%)	---
Transthoracic Echocardiography					
EF	69.5±5.27	68.75±4.34	70.63±3.04	68.71±7.65	.368
LVIDD	45.98±2.4 mm	46.25±2.27 mm	45.67±2.18 mm	46.19±2.82 mm	.671
LVIDS	27.82±2.49 mm	28.31±2.02 mm	27.26±1.68 mm	28.19±3.47 mm	.297
PWT	9.52±1.23 mm	9.06±1.12 mm	9.37±1.24 mm	10.05±1.16* mm	.037
IVST	13.9±12.67 mm	12.25±1.44 mm	16±19.44 mm	12.48±1.69 mm	.535
LA	36.15±3.3 mm	34.56±2.61 mm	35.89±2.99 mm	37.71±3.59* mm	.011
TAPSE	19.56±1.62 mm	20.69±1.35 mm	19.33±1.49 mm	19±1.61*# mm	.003
PVSP		29.88±3.44 mmHg	29.07±4.71 mmHg	37.48±4.97*# mmHg	.000

* indicates a statistically significant difference with mild OSA, # indicates a statistically significant difference with moderate OSA, one-way ANOVA followed by Duncan test.

BP: Blood pressure. HR: heart rate. RR: respiratory rate. SPO_2: oxygen saturation. RA: room air. EF: ejection fraction. LVIDD: left ventricular internal dimensions in diastole. LVIDS: left ventricular internal dimensions in systole. PWT: posterior wall thickness. IVST: interventricular septum thickness. LA: left atrium. TAPSE: Tricuspid annular plane systolic excursion. PVSP: pulmonary venous systolic pressure.

Table 3: Ambulatory BP monitoring (ABPM) findings and detection of masked hypertension

Ambulatory BP monitoring (ABPM) findings		
Overall average of systolic BP	149.1±7.8 mmHg	
Overall average of diastolic BP	95±5.9 mmHg	
Daytime average systolic BP	153.4±8.1 mmHg	
Daytime average diastolic BP	98.2±5.0 mmHg	
Nighttime average systolic BP	143.6±7.3 mmHg	
Nighttime average diastolic BP	92.3±4.9 mmHg	
Maximum systolic BP	164.3±9.6 mmHg	
Maximum diastolic BP	102.5±7.2 mmHg	
Minimum systolic BP	134.3±6.8 mmHg	
Minimum diastolic BP	87.9±5.8 mmHg	
Difference in detected hypertension		
Hypertension by ABPM 24h	57 (89.1%)	P value (McNemar Test) = 0.00
Hypertension (resting BP)	28 (43.7%)	
Difference in systolic BP		
Overall systolic BP	149.12±7.83	P value (paired sample t-test) = 0.001
Resting systolic BP	141.09±18.6	
Difference in diastolic BP		
Overall diastolic BP	95±5.93	P value (paired sample t-test) = 0.000
Resting diastolic BP	87.5±12.02	

Categorical data as Frequency and percent, McNemar Test and Continuous data were represented as mean and SD, paired sample t-test.
BP: blood pressure.

Table 4: Correlation between night-time average diastolic BP and Cardiovascular complications
AF: atrial fibrillation. LA: left atrium. IVST: interventricular septum thickness. PVCs: premature ventricular contractions.

Cardiovascular complications	R	P value
AF	-.050	.695
Overallischemia	-.083	.515
Abnormal LA	-.049	.701
Abnormal IVST	.365**	.003
Region alwallmotion abnormality	-.101	.427
Mild mitral regurgitation	-.141	.267
Mild tricuspid regurgitation	.043	.737
Diastolic Dysfunction	.434**	.000
infrequent PVCs	.0٦٢	.٦٣

Table 5: Distribution of overall detected cardio-vascular complications regarding grades of OSA
AF: atrial fibrillation. PWT: posterior wall thickness. IVST: interventricular septum thickness. LA: left atrium. PVCs: Premature ventricular contractions.

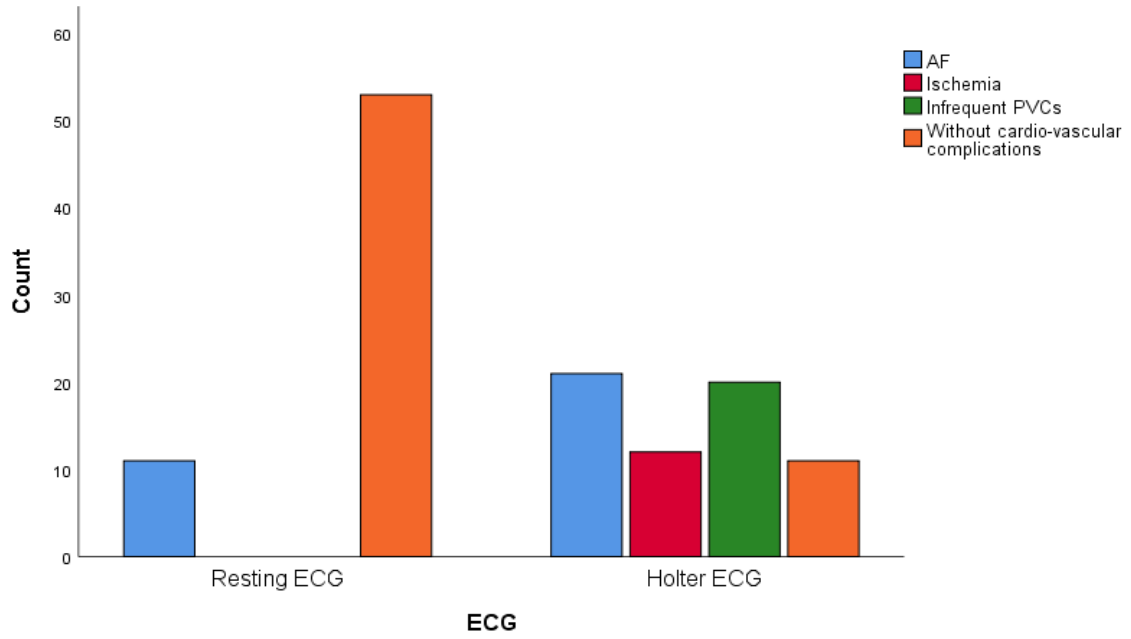

Figure 1: Bar chart for cardio-vascular complications detected by resting ECG and Holter ECG

DISCUSSION

Recurrent complete (apneas) and partial (hypopneas) upper airway obstructive episodes cause intermittent hypoxemia, autonomic instability, and fragmented sleep, which are the hallmarks of obstructive sleep apnea (OSA) [11]. In patients with OSA, a non-dipping nocturnal blood pressure profile is very likely [12].

Heart disease (CVD) continues to be a leading cause of morbidity and death. A growing understanding of cardiovascular disease (CVD) as a systemic process with multiple determinants has led to a greater emphasis on modifying cardiovascular risk factors for primary and secondary prevention, coinciding with the development of novel and enhanced therapies for established CVD such as heart failure or coronary artery disease [13].

Thus, in addition to Holter ECG and echocardiography, we used ABPM to diagnose diastolic blood pressure non-dipping and to evaluate the cardiovascular consequences in OSA patients. In this study, 64 OSA patients who were not dippers were enrolled. Of those with OSA, 32.8% had severe, 42.2% had moderate, and 25.0% had mild.

According to estimates, obstructive sleep apnea (OSA) affects 40% to 60% of patients with cardiovascular disease (CVD), as well as 34% of men and 17% of women in the general population. In addition, the prevalence is rising due to the aging population and the obesity epidemic, with these numbers showing a 30% increase over the preceding 20 years [13].

Our findings showed that the apnea-hypopnea index (AHI) and BMI were positively correlated. Compared to individuals with mild and moderate OSA, patients with severe OSA had greater BMIs. Severe OSA was present in all individuals with grade 3 obesity but not in any patient with grade 1 obesity.

Our research indicates that obesity is regarded

as a significant risk factor for the onset and advancement of OSA. Compared to adults of normal weight, the prevalence of OSA in obese or extremely obese patients is about twice as high [14].

Every patient in our study that is included has dyslipidemia. Increased levels of oxidative stress, lipid peroxidation, and increasing atherosclerosis have all been related to obstructive sleep apnea (OSA) [15]. Dyslipidemia is independently correlated with both moderate and severe obstructive sleep apnea (OSA) [16].

Twenty-eight patients (43.8%) of our study group were previously known hypertensive -with resting systolic blood pressure of mean 141 mmHg and SD of 18.6 mmHg, and resting diastolic blood pressure of mean 87.6 mmHg and SD of 12 mmHg-, but with the 24h ABPM there were 57 of them (89.1%) discovered hypertensive -with overall average systolic blood pressure of mean 149 mmHg and SD of 7.8 mmHg, overall diastolic blood pressure of mean 95 mmHg and standard deviation (SD) of 5.9 mmHg, Day-time average systolic BP of mean 153.4 mmHg and SD of 8.1 mmHg, day-time average diastolic BP of mean 98.2 mmHg and SD of 5 mmHg, night-time average Systolic BP of mean 143.6mm Hg and SD of 7.3 mmHg, and night-time average diastolic BP of 92.3 mmHg and SD of 4.9 mmHg denoting systolic and diastolic non-dipping of all patients-. This result is consistent with the findings of Tietjens et al., [13] and Logan et al., [17], who reported that 30% to 50% of hypertensive patients will also have concomitant OSA. OSA is a highly prevalent condition in these patients. Patients with resistant hypertension, of which up to 80% may develop OSA, are particularly affected by this.

According to our research, patients with severe OSA had greater blood pressure than those with mild or moderate instances. Sapina-Beltran et al. [18] discovered, in line with our findings, that people with mild to moderate or no OSA had lower blood pressure readings than patients with severe OSA. Consequently, they suggested that treating underlying causes of CVD, such as OSA, may be beneficial when trying to improve BP control, particularly at night, and may suggest novel therapeutic strategies in addition to pharmacotherapy.

Like our result, Sova et al., [19] examined the diagnosis rate of masked uncontrolled hypertension (MUCH) and masked hypertension (MH) in OSA patients using ambulatory blood pressure measurements (ABPM) and routine blood pressure measurements. The systolic and diastolic ABPM values were notably greater than those obtained from a standard blood pressure check. The non-dipping phenomenon was observed in 38 cases or 58.4%. Fifty-five patients (84.6%) had nocturnal hypertension. They concluded that even in cases where routine blood pressure readings are normal, a 24-hour ABPM is still required since patients with OSA had a significantly greater prevalence of Masked Hypertension/ Masked Un-Controlled Hypertension MH/MUCH.

Every patient in our study had an echocardiogram and every patient had diastolic dysfunction, IVST was elevated in 65.6%.

According to reports, periodic hypoxia may cause left ventricle (LV) remodeling, which was thought to be the root cause of LV dysfunction brought on by OSA. This finding is consistent with our research [20].

Bradley et al., [21] revealed that the production of negative intra-thoracic pressure

and the rise in systemic blood pressure as a result of hypoxia-induced sympathetic nervous system activation led to an increase in LV trans-mural pressure. According to Jean-Louis et al. [22] this causes an increase in afterload and a decrease in LV preload, which can have an immediate impact on the LV systolic function. A higher risk of cardiac ischemia and arrhythmias results from the combination of increased LV afterload and increased heart rate (HR), which also persistently leads to LV hypertrophy and failure.

In our study, estimated pulmonary artery systolic pressure (EPASP) was found to be higher in individuals with severe forms of OSA on echocardiography, with a mean EPASP of 37.5 mmHg and a standard deviation of 5 mmHg. In agreement with our study, Kholdani et al., [23] reported that in patients with severe PH attributable to another primary cause, coexisting OSA can exacerbate the disease process and increase mortality. The pulmonary hypertension (PH) related to OSA is generally mild in the absence of additional cardiopulmonary disease, with average mean pulmonary artery pressure between 25 and 30 mmHg and rarely exceeding 35 mmHg. In addition, Masa et al., [24] found that patients with obesity hypoventilation syndrome, which is distinguished by awake hypercapnia Partial pressure of carbon dioxide (PaCO2) (Paco2 >45 mm Hg) and obesity, frequently experience moderate to severe PH and are at a higher risk of unfavorable outcomes, such as cor-pulmonale and death, in contrast to isolated OSA.

In our study, the number of arrhythmias identified by Holter ECG was much higher than that of resting ECG. This could be explained by the Holter ECG's accuracy. When collecting occasional irregularities and detecting arrhythmias that might not be seen during a resting ECG, a Holter ECG is especially helpful. It offers a continuous recording of the electrical activity of the heart for a prolonged amount of time, up to 48 hours. The likelihood of finding fleeting anomalies that could arise during regular activities or sleep is increased by this extended observation [25].

After a 24-hour Holter ECG, it was found that 21 patients (32.8%) had AF, which indicated that 10 patients (15.6%) had intermittent Atrial fibrillation (AF). Of the study group, 11 patients (17.2%) had AF at their resting ECG. This is consistent with the discovery made by Mehra et al. [26] that in patients without any underlying cardiac conditions, OSA is an independent risk factor for AF.

It has not been conclusively established that OSA causes AF, yet both conditions are independently linked to similar unfavorable outcomes. Common risk factors for both conditions include obesity, advancing age, male sex, hypertension, and heart failure. In patients with OSA, there are multiple potential processes that could be the substrate and trigger of AF. Hypoxia and hypercapnia, changes in intrathoracic pressure, elevated sympathetic tone, and autonomic dysregulation are the outcomes of acute apneic episodes. Atrial fibrosis with downregulated connexin and electrophysiological abnormalities can result from both abrupt negative changes in intrathoracic pressure and chronic recurrence, which can also contribute to morphological and functional atrial remodeling [27].

In contrast to Mehra et al.'s study [26], which found that the main factors causing cardiac electrical abnormalities and raising the risk of arrhythmias were OSA-related hypoxia,

sympathovagal imbalance, and the mechanical effects of negative intrathoracic pressure on the ventricular free walls, we also observed infrequent extrasystoles in the Holter ECGs of 20 patients (31.2%) of our studied group. No other complex ventricular arrhythmias were found in our study.

The results from Shah et al., [28], which stated that OSA has also been implicated in coronary artery calcification, plaque instability, and plaque vulnerability and has been associated with a 2-fold increase in risk of cardiovascular events or death, were also aligned with the observation that twelve of our patients (18.7%) had ECG ischemic changes in the 24-hour Holter ECG that were not noted in the regular resting ECG of the same patients.

The results from Bouloukaki et al. [29] showed that CRP is one component of the underlying inflammatory process in OSA, and there was a positive connection between the apnea-hypopnea index (AHI) and CRP. Additionally, the findings from Yi et al. [30] showed a correlation between increased CRP and the severity of OSA. Additionally, increased CRP showed a potential causal relationship with OSA. Additionally, OSA patients have higher levels of inflammatory mediators, such as CRP, which do not seem to be correlated with polysomnographic parameters but do positively correlate with the degree of obesity, according to a study by Wali et al. [31].

Furthermore, a positive correlation was seen between AHI and LDL, TG, and cholesterol. Seifen et al. [32] and Karkinski et al. [33] reported that obesity and OSA are significant risk factors for dyslipidemias. In people who are not obese, OSA may significantly contribute to a deterioration of lipid metabolism. However, compared to non-obese patients, the role of OSA is less significant in obese patients because of the metabolic alterations brought about by their excess weight.

Conclusions:

ABPM 24h is considered an effective method for identifying concealed hypertension in patients with non-dipper OSA in both the systolic and diastolic forms. There is an increased incidence of OSA of CVD that increases with increasing degree of OSA. So we recommend periodic cardiovascular examinations for patients suffering from OSA.

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Conflict of interest

The authors declare that they have no conflicts of interestwith respect to authorship or publication of this article

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