Original Article

Obstructive Sleep Apnea and Blood Pressure

Obstructive Sleep Apnea and Blood Pressure: A Cross Sectional Study in Our Local Population

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ABSTRACT

Objective: To evaluate the association of obstructive sleep apnea with blood pressure in our local population.
Study Design: Cross sectional study
Place and Duration of Study: This study was conducted at the Sleep Laboratory, Polysomnography Dept., Dow University Hospital, Karachi from December 2013 to June 2015.
Materials and Methods: Total 100 subjects were evaluated for study, in which 50 were OSA cases while 50 were control subjects. All subjects were went through informed consent, detailed history, physical examination and ESS score calculation. OSA subjects were examined by overnight polysomnography to confirm diagnosis of OSA and to divide them into groups according to severity of OSA. Systolic and Diastolic blood pressure were evaluated and analyzed by using SPSS version 20.
Results: Mean systolic blood pressure (mm Hg) was significantly higher in cases as compared to the controls (138.60 ± 14.07vs 129.40 ±14.93) ($p < 0.002$). Frequency of personswith high blood pressure was 74% in cases and 30% in controls. Frequency of persons with highblood pressure was also associated with severity of apnea as it was 30% in control subjects while it was 58.3%, 76.5% and 81.0% in mild apnea, moderate apnea and severe apnea groups respectively.
Conclusion: Study findings are suggesting that obstructive sleep apnea is associated with higher blood pressure; it is also associated with severity of OSA in our population.Our study is emphasizing the need of improving our understanding regarding thepathophysiology of OSA and its relation with hypertension.
Key Words: Obstructive sleep apnea , sleep , Blood pressure

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INTRODUCTION

Proper sleep in necessary for a healthy and active life. Inadequate sleep causes daytime sleepiness which can be an antecedent to falls, declining quality of life, concentration failure, morning headaches, loss of energy, irritability and fatigue. Inadequate sleep is associated with multiple neurological and metabolic abnormalities which can not only effect quality of life but can also shorten life span[1,2].

Sleep related respiratory disorders (SRBDs) are a wide group of disorders. Some of them are related to habitual snoring while some are related to partial or complete pause in breathing and decrease oxygen saturation during sleep.

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Out of all SRBDs, obstructive sleep apnea (OSA) is a condition which required early diagnosis and treatment as it is proven to be related with multiple neurological and metabolic disturbances in the body[3,4]. OSA is a condition in which there are repeated events of complete or partial upper airway obstruction during sleep, that last for more than 10 seconds, associated with decrease oxygen saturation and excessive day time sleepiness (EDS). Severity of the disorder is characterized by the frequency of apneas and hypopnea episodes per hour of sleep which is termed as Apnea-Hypopnea Index (AHI). OSA is a relatively common sleep disorder which if left untreated leads to significant mental and physical problems[5].

According to studies OSA is a cause of disruptive snoring, frequent arousals during sleep, excessive daytime sleepiness, impaired work performance, cognitive dysfunction, decreased mental alertness, attention deficit, memory problems, anxiety, displeasure or depression, lack of energy and tiredness, decrease in health related quality of life, gastro-esophageal reflux, obesity, diabetes, stroke and many cardio-vascular disorders[6,7].

Events of repetitive respiratory collapse in OSA impose substantial adverse effects on multiple organ systems. Over the past two decades, the pathophysiological

mechanism of OSA has been better understood, but exact contributory pathways are still not clearly described. Studies suggested the involvement of multi-factorial mechanism in the association of OSA with metabolic disturbance and cardio-vascular risk in relation to OSA.

According to many studies conducted in multiple countries OSA patients show a strong association with systemic hypertension. Studies have suggested not a single pathway, but rather a combination of mechanisms that could lead to hypertension risk in OSA patients[4].

Extensive data is available to understand the alteration inautonomic nervous system (ANS) that occur with the transition from wakefulness to sleep, and throughout sleep state dynamics. Blood pressure(B.P) and heart rate (H.R) decreases during deep NREM sleep than when awake due to elevated parasympathetic tone and sympathetic withdrawal. During transitioning phase from NREM sleep to REM sleep, BP and HR increase and become more unstable. In REM sleep there is an increase in sympathetic nervous system activity (SNA) above the levels found during wakefulness. Considering the close relationships between sleep regulatory mechanisms and ANS it is logical to expect that, any sleep disturbance can theoretically lead to alterations in sympathetic activity, and may thus disturb blood pressure regulatory system. Repetitive episodes of intermittent hypoxia (IH) and CO_2retention canalso augment SNA via stimulation of central and peripheral chemoreceptors[3,8]. The prominent role of hypoxia in promoting an increase in BP in both animal model and human has been demonstrated. Elevated BP found to be associated with increased chemo reflex whereas barore flex, which is protective for the vascular wall, decreases in OSA[9].

MATERIALS AND METHODS

Total 100 subjects were evaluated for study, in which 50 were OSA cases while 50 were control subjects. All subjects were went through informed consent, detailed history, physical examination and Epworth sleep scale (ESS) score calculation to evaluate their day time sleepiness and chances of having OSA.

Suspected case subjects were initially scrutinized from sleep clinics. They were with multiple sleep related complains e.g. sleep disturbance, loud snoring, day time sleepiness, frequent arousal from sleep, morning headache etc. ESS score more than 9 indicates day time sleepiness. Suspected OSA subjects were examined by overnight polysomnography to confirm diagnosis of OSA and to divide them into groups according to severity of OSA based on their AHI. As mild (AHI = 5-15), moderate (AHI= 15-30) and (AHI <30)[10]. A Polysomnography (PSG) is a comprehensive multi-channel recording of the biophysiological changes that occur during sleep[11]. Age, gender and BMI matched controls subjectswere selected without any sleep related complain and ESS score less than 9. Systolic and Diastolic blood pressure were evaluated and analyzed by using SPSS version 20. Subjects with BP ≥ 130/85 mmHg or already on hypertensive medicines were consider with high blood pressure.

RESULTS

The research sample comprised of 100 individuals, 50 each with and without OSA, the later served as controls. Mean age and BMI was not significantly different in both groups. The mean AHI for cases was 32.795 ± 22.70 while it was assumed as Zero for controls.

Mean systolic blood pressure (mm Hg) was significantly higher in cases as compared to the controls (138.60 ± 14.07 vs 129.40 ±14.93) ($p < 0.002$). Although diastolic blood pressure was also higher in OSA group as compared to controls (85.84 ± 13.83 vs 80.90 ± 14.20) but the difference in diastolic blood pressure was not statistically significant ($p < 0.081$) in both groups. The data is shown in table 1.

Hypertension was found to be significantly ($p < 0.000$) more common in patients with OSA than in controls (table 2).

Table No.1: Comparison of mean blood pressure among subjects with and without Obstructive Sleep Apnea

Mean Blood pressure	Controls	Cases	p-value
Systolic Blood Pressure (mmHg)	129.40 (125.16-133.64)	138.60 (134.60-140.60)	0.002*
Diastolic Blood Pressure (mmHg)	80.90 (76.86-84.94)	85.84 (81.91-89.77)	0.081

*Statistically significant difference, p-values generated by t-test

Table No.2: Frequency Distribution of Normal and High blood pressure Subjects in Cases and Control (n=100)

Categories with normal and high B.P	Control n (%)	Case n (%)	P Value
Normal BP/No Anti-hypertensive medicine	35 (70%)	13 (26%)	.000*
High BP /Anti-hypertensive medicine	15 (30%)	37 (74%)	

*Statistically significant difference, p-values generated by t-test

The % of subjects who were not the diagnosed cases of hypertension and were not on antihypertensive medicine but with normal blood pressure was 26% (n=13) in cases whereas they were 70 % (n=35) in controls. Likewise the % of subjects with diagnosed hypertension or with high blood pressure was 74% (n=37) in cases and 30% (n=15) in controls. The data

documents that % population of OSA group with high blood pressure was almost double as compared to the controls.

Hypertension was also found to be associated with the severity of apnea; increase in the severity of apnea, increases % of population with high blood pressure. It was 30% in control subjects while it was 58.3%, 76.5% and 81.0% in mild apnea, moderate apnea and severe apnea groups respectively (Table:3) (Figure.1)

Table No.3: Number and percentage of subjects in mild, moderate and severe OSA groups with normal and high blood pressure

Categories with normal and high B.P	OSA CASES			Total In Case group
	Mild Apnea (AHI=5-15) n(%)	Moderate Apnea (AHI=15-30) n(%)	Severe Apnea (AHI >30) n(%)	
Normal B.P /No Antihypertensive Medicine	5 (41.7%)	4 (23.5%)	4 (19%)	13 (26%)
High BP /Anti-hypertensive Medicine	7 (58.3%)	13 (76.5%)	17 (81%)	37 (74%)
Total	12 (100%)	17 (100%)	21 (100%)	50 (100%)

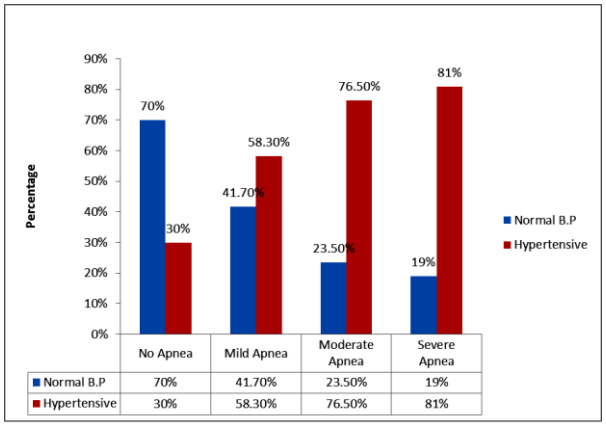

Figure No.1: Association of blood pressure with severity of apnea

DISCUSSION

Our study is in line with previous studies showed increase frequency of hypertension in OSA patients as compare to controls. We have included equal number of male and female participants in both groups. They were matched with age as well as with their BMI, so any chance of biasnessin our results due to age, gender or BMI was eliminated.

According to studies Sleep heart health studies (SHHS) has described OSA as an important and self-sufficient causative factor for hypertension[12]. In present study hypertension was documented with significantly higher frequency in OSA patients and revealed direct relationship between these two, independent of age and obesity. These findings are also support the previous studies showing strong and direct association of OSA with systemic hypertension[4]. Some previous studies have shown the prevalence of hypertension to be almost twice in OSA group compared to controls (91.3% vs. 57.1%; p=0.041)[13]. In present study mean systolic blood pressure (mm Hg) was much higher in patients with OSA as compared to control (138.60 vs.129) with P value = 0.002. However mean diastolic blood pressure did not reflect statistically significant difference. In the present study significant positive correlation between hypertension and severity of apnea has been documented.

Wisconsin Sleep Cohort Study after four years observation has provided the most compelling data indicating the causative role of apnea in the pathogenesis of hypertension and indicates that an AHI of 15 and more heightened the possibility of having hypertension by 80%[14].

A series of cross-sectional analyses were carried out demonstrating that an increased AHI was related to high chances of having hypertension; and this relationship remained prominent even after correcting for confounding variables like age and obesity. This assumption has been supported by observations from interventional studies, which showed that treatment with CPAP may decrease both systolic and diastolic blood pressures[15,16]. All these findings support the suggestion that OSA is likely to be a risk factor for hypertension and subsequent cardio vascular morbidity. The present study on Pakistani population thus supports this suggestion. Evidences suggest that OSA could be a modifiable risk in reducing overall prevalence of hypertension in our Pakistani population. Better understanding of OSA and its relationship with hypertension could help to reduce the burden of this disease.

Studies considered OSA as an etiologic factor for the development of hypertension as well as for resistant hypertension. One third of hypertensive population is suffering from OSA[4]. The `seventh report` issued by "Joint National Committee" has recognized OSA as a significant cause of hypertension[17]. Expert panels have recommended that OSA and its treatment should be a part of management of patients with resistant hypertension[18]. OSA-mediated sympathetic activation is known to promote the development of resistance hypertension.

The relationship of apnea and hypertension has been reviewed extensively and different factors resulting in hypertension in OSA patients been reported: increased sympathetic activity, systemic inflammation, renin-angiotensin-aldosterone system dysfunction, increased vascular resistance, oxidative stress, damage to endothelial function, metabolic dysregulation, atherosclerosis and vascular injury[17,19] . So tonic elevation of sympathetic neural activity due to sleep fragmentation and intermittent hypoxia and disturbance in sleep related baroreceptor activity could be considered as most important factors.

Awareness and understanding of the etiologic mechanisms underlying hypertension in OSA would assist us in the development of therapies against this co morbidity. Enough and proper sleep is important to reduce chances of hypertension. Almost1400 years ago, Muhammad (Peace Be Upon Him) explained the value of proper sleep for good health. The Prophet (PBUH) said to one of his companions (IbnAmr) who was praying the whole night "Offer prayers and also sleep at night, as your body has a right on you" (SB 1874)[20]. Which indicate importance of proper sleep from Islamic point of view.

CONCLUSION

There is a need to recognize importance of proper sleep at night and any sleep disturbance should be considered for immediate and proper therapy to prevent co-morbidities like hypertension. Our study is emphasizing the need of improving our understanding regarding the pathophysiology of OSA and its relation with hypertension.

Author's Contribution:

Concept & Design of Study:	Ambreen Qamar
Drafting:	Hafiz Syed Imran-Ul-Haq
Data Analysis:	Rashid Ahmed, Afsheen Qamar
Revisiting Critically:	Ambreen Qamar, Hafiz Syed Imran-Ul-Haq
Final Approval of version:	Ambreen Qamar

Conflict of Interest: The study has no conflict of interest to declare by any author.

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