ISSN 2277-7695

Online Available at www.thepharmajournal.com

THE PHARMA INNOVATION

Prevalence and etiological outcome of hypertension in patients with obstructive sleep apnea hypopnea syndrome

Ashok Kumar Garg

Associate Professor, Department of General Medicine, National Institute of medical science and research Jaipur Rajasthan, India

Introduction: The aim of the study was to determine the Etiological outcome and pervasiveness of Hypertension in Patients with Obstructive Sleep Apnea Hypopnea Syndrome.

Materials and Methods: One nurse completed the study questionnaire, When each patient was recruited in our sleep center, the nurse gathered his or her demographic information, which included name, gender, age, job, height, weight, family history, smoking and drinking history, snoring history, sleepiness tendency in different conditions, hypertension history, and antihypertensive drug application. Next, the body mass index (BMI = weight in kilograms divided by height in meters squared) and Epworth sleepiness score of each participant were calculated. BP were taken and charted of morning evening and night. The subjects were divided into four groups based on the PSG results: the con-trol group (control, n = 275) mild OSAHS (mild, n = 430) moderate OSAHS (mod- erate, n = 650) and severe OSAHS (severe, n = 870).

Result: Increased AHI values and decreased $LSaO_2$ values were associated with increased daytime, evening, nighttime, and morning MBPs in all groups, after controlling for related confounding variables. Patients with OSAHS have high levels of sympathetic nerve activity.

Conclusion: OSAS is associated with hyper-tension and hypertension associated end-stage organ diseases such as stroke, coronary heart disease, and arrhythmia, the employment of CPAP is highly encouraged as CPAP therapy seems to assist blood pressure control at least in those with severe apnea, resistant hypertension, and daytime sleepiness.

Keyword: Etiological, pervasiveness, Hypertension, Obstructive Sleep Apnea, Hypopnea Syndrome

Introduction

The severity of OSA is determined by the apnea hypopnea index (AHI) which is a measure of the number of periods of obstructed breathing per hour of sleep. The prevalence of OSA (AHI. 5 events/hour) with associated daytime sleepiness has been estimated at between 2% and 7% in diverse middle aged adult populations, [1] whilst the prevalence of OSA (AHI. 1 event/hour) in children is between 1%–3% [2]. Adults with OSA are typically centrally obese, and although this obesity is strongly causally linked to the condition, there is an increased prevalence of cardiovascular morbidity and mortality amongst OSA sufferers above what would be expected from obesity alone. In addition to OSA, patients often present with one or more comorbidities including dyslipidemia, glucose intolerance, and hypertension. Obstructive sleep apnea hypopnea syndrome (OSAHS) is a common sleep-related breathing dis- order, which is characterized as frequent upper airway col- lapse and obstruction [3]. He *et al.* showed an independent and definite association between OSAHS and hypertension [4]. Both OSAHS and hypertension are chronic

conditions that, if untreated, are associated with consequent impairment of life quality and considerable morbidity and mortality resulting from chronic conditions that affect cardiopulmonary function and acute cardiovascular events that may end in nocturnal sudden death [5]. Thus, expanding our understanding of the prevalence and clinical features of hypertension in patients with OSAHS and, thereby, potentially promoting more effective treatment and nursing interventions are essential to improve the long-term clinical management of this disorder. Our understanding of OSAS has evolved from initially regarding it as merely an annoying social situation to the recognition that OSAS may act through various mechanisms to increase cardiovascular risk and lead to increased morbidity and mortality [6]. In particular, there is evidence that untreated OSAS may contribute to the pathophysiological mechanisms underlying the origin and/or development of hypertension, cardiac ischemia, myocardial infarction, congestive heart failure, and stroke [7]. Of the different possible consequences of OSAS in patients, the most widely recognized may be the development of systemic hypertension. While many reviews have described the association between OSAS and hypertension, the diagnosis, prevalence, etiology, and new mechanisms linking OSAS to hypertension are outlined in this study.

Corresponding Author's Contact information:
Ashok Kumar Garg *
Associate Professor, Department of General Medicine, National Institute of medical science and research Jaipur Rajasthan, India
E-mail: hkps0320@gmail.com

Material and Methods

2225 consecutive outpatients (aged 18Y-81 years) who came to the sleep center of the National Institute of medical science and research Jaipur Rajasthan. Were recruited between March 2015 and May 2018. A trained nurse explained the study protocol to each participant and obtained his or her informed consent. One nurse completed the study questionnaire, When each patient was recruited in our sleep center, the nurse gathered his or her demographic information, which included name, gender, age, job, height, weight, family history, smoking and drinking history, snoring history, sleepiness tendency in different conditions, hypertension history, and antihypertensive drug application. Next, the body mass index (BMI = weight in kilograms divided by height in meters squared) and Epworth sleepiness score of each participant were calculated. BP were taken and charted of morning evening and night. The subjects were divided into four groups based on the PSG results: the control group (control, n = 275) mild OSAHS (mild, n = 430) moderate OSAHS (moderate, n = 650) and severe OSAHS (severe, n = 870).

Three consecutive BP values were read on the right arm in the supine position at 15-second intervals after 5 minutes of rest at each time point. The average of the three consecutive BP values was adopted and used in the analysis. According to the guidelines, hypertension was defined as systolic BP of at least 140 mmHg, diastolic BP of at least 90 mmHg during the daytime BP, and/or existing hypertensive history [8]. All participants performed a nocturnal PSG to measure oral and nasal airflow, respiratory effort, snoring, heart rate, and oxygen saturation (SaO_2). Severity of OSAHS was assessed using the apnea Hypopnea index (AHI). Statistical analysis and chart were obtained from the SPSS 18.0 software package (SPSS, Inc., Chicago, IL, USA).

Result

Table 1: Demographic Data and the Prevalence of Hypertension by Group

Variable	Control (n = 275)	Mild (n = 430)	Moderate (n = 650)	Severe (n = 870)
Gender (male/female)	160/115	350/80	578/72	729/141
Smoking/nonsmoking (n)	126/149	220/210	335/315	465/405
Drinking/nondrinking (n)	215/60	312/118	440/210	250/620

Age (years)	41.23 ± 21.43	46.32 ± 12.28	42.15 ± 9.22	44.6 ± 12.34
BMI (kg/m^2)	24.15 ±8.14	27.44 ± 5.35	26.39 ±5.82	28.78 ± 3.21
AHI (episodes/hour)	3.29 ± 2.92	10.16 ±6.82	19.10 ±3.70	43.11 ± 18.86
$LSaO_2$ (%)	82.52 ±7.73	79.35 ±16.12	73.40 ±7.09	59.95 ±15.95
ESS (points)	6.07 ±35.73	8.69 ±2.78	9.12 ±3.67	10.61 ±4.83
Prevalence of hypertension (%)	19.82	37.43	40.11	52.77

Table 2: The Daytime, Evening, Nighttime and Morning MBPs in Different Group

Variable	Control M ± SD	Mean M ± SD	Moderate M ± SD	Severe M ± SD	F
Daytime MBP (mmHg)	91.76±12.45	95.23±14.52	96.52±10.13	99.34±21.43	82.32
Evening MBP (mmHg)	91.06±11.82	94.20±9.46	96.76±21.9	99.57±17.34	86.9
Nighttime MBP (mmHg)	88.34±12.89	92.78±14.65[a]	95.67±14.23[ab]	99.99±13.45[abc]	106.94
Morning MBP (mmHg)	91.73±11.82	95.14±12.78[a]	97.45±12.58[ab]	103.55±13.96[abc]	122.54
Rhyme nighttime/daytime	0.989±0.091	0.994±0.086	1.007±0.089[a]	1.014±0.109[ab]	8.91
Rhyme nighttime/daytime	1.025±0.112	1.014±0.099	1.023±0.091	1.045±0.106[abc]	20.3

Discussion

AHI and $LSaO_2$ were closely related to the BP values in the four groups that were examined in this study. Increased AHI values and decreased $LSaO_2$ values were associated with increased daytime, evening, nighttime, and morning MBPs in all groups, after controlling for related confounding variables. Patients with OSAHS have high levels of sympathetic nerve activity. Recurrent episodes of airway obstruction result in hypoxia and hypercapnia, which increases sympathetic neural tone through chemoreceptor reflexes and other mechanisms and, in turn, causes vasoconstriction and marked increases in BP [9]. Furthermore, high levels of endothelia and Angiotensin II [10] as well as of endothelial dysfunction [11] induced by systemic inflammation and oxidative stress may contribute to increased BP. A recent study revealed that among apparently normotensive male OSAS patients, masked hypertension is present in one-third of patients and there is a progressive impairment of arterial stiffness in OSAS patients with masked hypertension, indicating that the diagnosis of masked hypertension may be underestimated in OSAS patients and that OSAS has an association with arterial stiffness independent of masked hypertension [12]. Moreover, obesity may be involved in the relationship between OSAS and masked hypertension. Although common etiologies of masked hypertension include stressful living situations, sleep apnea resulting from obesity is also a possible etiology [13]. In general, OSAHS not only increases BP value but also changes the BP CR. A recent study showed that normotensive persons with a no dipping BP profile experienced increased target organ damage [14]. This study provides a new view on treatment options for the appropriate management of hypertension in patients with OSAHS. Treatments for OSAHS, if present, may decrease BP value and improve circadian BP rhythm, which contribute to reducing hypertension-related target organ damage and de- creasing morbidity and mortality in patients with hypertension. This also reminds nurses to pay attention to circadian BP variations in patients with OSAHS, especially in those un-diagnosed hypertensive patients who exhibit normal daytime BP but abnormal nighttime BP [15]. Use of ambulatory BP monitoring, as suggested in recent guidelines [16] may further increase the ability to monitor BP regularly in subjects with OSAHS. Therefore, nurses in sleep centers have plenty of work to do. Cheng, *et al.* found that the behavior of patients and a chronotherapy intervention increased the drop in patients' nocturnal BP, in- creased the number of patients with dipper and decreased the number of patients with reverse dipper, and improved the BP surge in the morning [17].

Conclusions

Over the last decade, there has been increased interest in OSAS-related research and increased understanding of the OSAS related

cardiovascular complications. Because OSAS is associated with hyper-tension and hypertension associated end-stage organ diseases such as stroke, coronary heart disease, and arrhythmia, the employment of CPAP is highly encouraged as CPAP therapy seems to assist blood pressure control at least in those with severe apnea, resistant hypertension, and daytime sleepiness. This field continues to be updated monthly. A number of key questions, however, remains to be resolved. More clinical research is warranted to characterize more fully the underlying mechanisms and to develop practicable strategies for OSAS treatment.

References

1. Punjabi NM, Caffo BS, Goodwin JL, *et al.* Sleep-disordered breathing and mortality: a prospective cohort study PLoS Med. 2009;6(8):e1000132.
2. Ebert CS, Drake AF. The impact of sleep-disordered breathing on cognition and behavior in children: A review and meta-synthesis of the literature. Otolaryngol Head Neck Surg. 2004;131(6):814–826.
3. Feng J, Chen BY. Prevalence and incidence of hyper- tension in obstructive sleep apnea patients and the relationship between obstructive sleep apnea and its confounders. Chinese Medical Journal, 1464Y1468. (Original work published in Chinese). 2009;122:12.
4. He QY, Feng J, Zhang XL, Liang ZA, Huang SG, Kang J, Chen BY. Elevated nocturnal and morning blood pressure in patients with obstructive sleep apnea syndrome. Chinese Medical Journal, (Original work published in Chinese). 125(10):1740Y1746.
5. Goodson BL, Wung SF, Archbold KH. Obstructive sleep apnea hypopnea syndrome and metabolic syndrome: A synergistic cardiovascular risk factor. Journal of the American Academy of Nurse Practitioners. 24(12):695Y703. DOI:10.1111/ j.1745-7599.2012.00771.x
6. Lanfranchi P, Somers VK. Obstructive sleep apnea and vascular disease. Respired Res. 2001;2:315–19.
7. Kohler M, Stradling JR. Mechanisms of vascular damage in obstructive sleep apnea. Nat Rev Cardiol. 2010;7:677–85.
8. Mancia G, Fagard R, Narkiewicz K, Redo´ n J, Zanchetti A, Bo¨ hm M. Task Force Members. ESH/ESC Guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension for the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). Journal of Hypertension. 31(7):1281Y1357. DOI: 10.1097/01.hjh.0000431740.32696.cc
9. Wolf J, Hering D, Narkiewicz K. Non-dipping pattern of hypertension and obstructive sleep apnea syndrome. Hyper-tension Research. 2010;33(9):867Y871. DOI: 10.1038/hr.2010.153.
10. Belaidi E, Joyeux-Faure M, Ribuot C, Launois SH, Levy P & Godin-Ribuot D. Major role for hypoxia inducible factor-1 and the endothelia system in promoting myocardial infarction and hypertension in an animal model of obstructive sleep apnea. Journal of the American College of Cardiology. 2009;53(15):1309Y1317. DOI: 10.1016/j.jacc.2008.12.050.
11. Bosc LV, Resta T, Walker B & Kanagy NL. Mechanisms of intermittent hypoxia induced hypertension. Journal of Cellular and Molecular Medicine. 2010;14(1Y2):3Y17. DOI: 10.1111/j.1582-4934. 2009.00929.x
12. Drager LF, Diegues-Silva L, Diniz PM, Bortolotto LA, Pedrosa RP, Couto RB, *et al.* Obstructive sleep apnea, masked hypertension and arterial stiffness in men. Am J Hypertens. 2010;23:249–54.
13. Mak RH, Bakris G. Pediatrics: masked hypertension: a risk factor in children with CKD. Nat Rev Nephrol. 2010;6:132–4.
14. Hermida RC, Ayala DE, Mojon A & Fernandez JR. Blunted sleep-time relative blood pressure decline increases cardiovascular risk independent of blood

pressure level. The ‘‘Normotensive Non-Dipper’’ paradox Chronobiology International, 30(1Y2):87Y98.
DOI: 10.3109/07420528.2012.701127

15. Parati G, Lombardi C, Hedner J, Bonsignore MR, Grote L, Tkacova R, I EU Cost Action B26 Members. Recommendations for the management of patients with obstructive sleep apnea and hypertension. European Respiratory Journal. 41(3):523Y538.
DOI: 10.1183/09031936.00226711
16. Mancia G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, Bohm M. Task Force Members. ESH/ESC Guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension for the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). Journal of Hypertension. 31(7):1281Y1357.
DOI: 10.1097/01.hjh.0000431740.32696.cc
17. Cheng M, Cheng SL, Zhang Q, Jiang H, Cong JY, Zang XY, *et al.* The effect of continuous nursing intervention guided by chronotherapeutics on ambulatory blood pressure of older hypertensive patients in the community. Journal of Clinical Nursing. 23(15Y16):2247Y2254.
DOI: 10.1111/jocn.12502