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Sudarshan G
Post Graduate, General Medicine, Rajah Muthiah Medical College, Chidambaram, Tamil Nadu, India

Umarani R
Professor, General Medicine, Rajah Muthiah Medical College, Chidambaram, Tamil Nadu, India

Naanjil Kumaran A
Assistant Professor, General Medicine, Rajah Muthiah Medical College, Chidambaram, Tamil Nadu, India

Corresponding Author:
Sudarshan G
Post Graduate, General Medicine, Rajah Muthiah Medical College, Chidambaram, Tamil Nadu, India

Prevalence of pulmonary hypertension in OSA patients with obstructive airway diseases

Sudarshan G, Umarani R and Naanjil Kumaran A

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Abstract
Objectives: The aim of this study is to study the prevalence of pulmonary hypertension in obstructive sleep apnea patients with obstructive airway disease using ECHO.
Subjects and method: The study was performed on 50 obstructive airway diseases (OAD) patients out of which 35 were associated with OSA. After getting informed consent, patients basic demographic details, clinical history, detailed general and systemic examination, spirometry, sleep study, Echo were performed for each patients.
Results: In our study pulmonary hypertension were higher in number in OSA patients compared to the NON-OSA patients. Among the obese patients higher number of pulmonary hypertension were observed. As far as severity is concerned more number of severe pulomonary hypertension were noted in OSA and Obese patients.
Conclusion: Pulmonary hypertension is invariably present in most of the OSA patients with obstructive airway diseases. Hence control of OSA by CPAP therapy in OAD patients will prevent or delay the development of pulmonary hypertension.

Keywords: obstructive sleep apnea (OSA), obstructive airway disease (OAD), pulmonary hypertension (PHTN)

Introduction
Obstructive sleep apnea is characterized by repeated episodes of upper airway occlusion that result in brief periods of breathing cessation (apnea) or a marked reduction in flow (hypopnea) during sleep. This pattern is accompanied by oxyhemoglobin desaturation, persistent inspiratory efforts against the occluded airway, and arousal from sleep [1].Chronic obstructive pulmonary disease (COPD), a common preventable and treatable disease, is characterized by persistent airflow limitation that is usually progressive and that is caused by an enhanced chronic inflammatory response in the airways and the lung to noxious particles or gases, smoking being the primary risk factor for the development of COPD [2]. Chronic obstructive pulmonary disease and sleep apnea each pose specific health risks on their own, but together they can be even more dangerous [3]. They are both serious, potentially life threatening conditions. Due to the breathing difficulties, those with COPD commonly have low oxygen levels at all hours of the day, and those who have both COPD and sleep apnea are at greater risk of prolonged oxygen desaturation at night than those with sleep apnea but without COPD [4]. Therefore, patients with overlap syndrome are particularly prone to the complications of chronic hypoxemia, such as cor-pulmonale, high blood pressure and heart disease. Bhavneesh Sharma *et al.* [5] conducted a study on effect of Pulmonary Function in Obstructive Airway Disease and Obstructive Sleep Apnea. Raj Kumar *et al.* [6] studied the risk of obstructive sleep apnea among middle aged chronic obstructive pulmonary disease and asthma patients by a home based sleep study and its association with atopy. Wan-Lu Sun *et al.* [7] studied about the impact of obstructive sleep apnea on pulmonary hypertension in patients with chronic obstructive pulmonary disease. Abou Shehata *et al.* [8] studied to investigate whether OSA by itself without any other cardiac or lung disease can lead to pulmonary hypertension, and to assess the effect of CPAP therapy on pulmonary artery pressure. In this study, the pulmonary hypertension between OSA and NON - OSA patients, obese and non- obese patients were observed for prevalence of pulmonary hypertension.

Aim of the Work

The aim of this study is to study the prevalence of pulmonary hypertension in these patients using ECHO.

Subjects and methods

The study was performed on 50 OAD patients out of which 35 were associated with OSA. After getting informed consent, patients basic demographic details, clinical history, detailed general and systemic examination, spirometry, sleep study, Echo were performed for each patients.

Materials and Methodology

Type of study : cross sectional
Sample size : 50
Time frame : 3 years

Inclusion Criteria

- Age>18 years
- COPD patients
- Asthma patients
- Obese patients

Exclusion Criteria

- Age<18 years
- Left side heart failure
- Recent use of opiods and sedatives
- Steroid use
- Thyroid disease
- Pregnant woman
- Stroke patients
- Upper airway obstruction like Adenoids

Result and Discussion

The prevalence of PHTN in OSA patients were showed in Table 1 and figure 2. Among 35 OSA patients 25 had pulmonary hypertension.

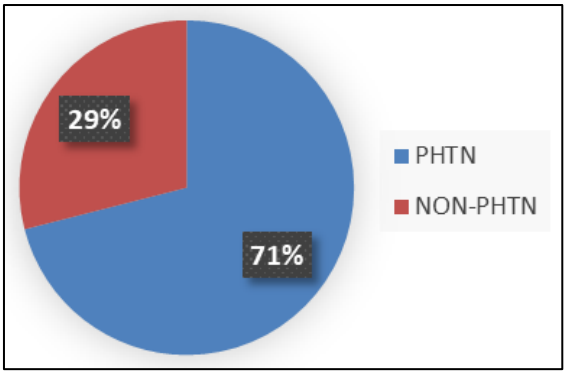

Fig 1: Prevelance of PHTN in OSA Patients

The pulmonary hypertension between OSA and NON OSA among OAD patients were showed in Table 2 and figure 2. In this pulmonary hypertension were higher in number in OSA compared to NON OSA patients. Odd ratio, 95% CI and p=value were calculated; Odds ratio is 16.25; 95% CI is 3.0911 to 85.4260; p-value is 0.001< 0.05 statistically significant.

Table 1: Prevelance of PHTN In OSA patients

PHTN	OSA(n=35)	Percentage
YES	25	71%
NO	10	29%

Table 2: PHTN in OSA and NON-OSA among OAD patients

Pulmonary HTN	OSA(n=35)	NON-OSA(n=15)
YES	25	2
NO	10	13
Odds ratio	16.25	
95% CI	3.0911 to 85.4260	
P Value	0.001	

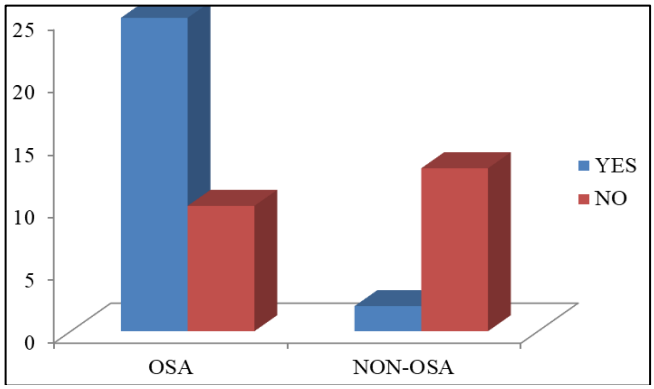

Fig 2: PHTN in OSA and NON-OSA among OAD patients.

The pulmonary hypertension among obese and non-obese patients with OAD were showed in table 3 and graphically represented in figure 3.Among the obese patients more number of pulmonary hypertension were observed. Odds ratio is 3.8857, 95% CI is 1.1906 to 12.6811, P value is 0.0245 <0.05 statistically significant

Table 3: PHTN in obese and non obese among OAD patients of BMI>25

Pulmonary HTN	OBESE	NON-OBESE
Yes	17	10
No	7	16
Odds ratio	3.8857	
95 % CI	1.1906 to 12.6811	
P Value	0.0245	

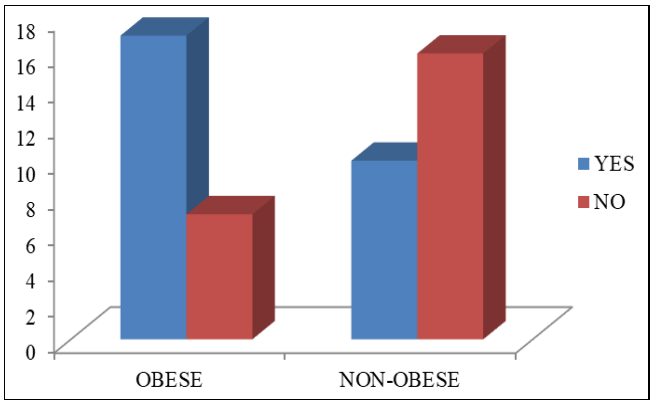

Fig 3: PHTN in obese and non obese among OAD patients of BMI>25

Table 4 and figure 4 showed prevalence of PHTN in OSA patients with obesity. In this study we found 65% of OSA patients with obesity had PHTN

Table 4: Prevalence of PHTN in OSA patients with obesity (BMI>25)

Pulmonary HTN	OBESE(n=17)	Percentage (%)
OSA-YES	11	64.7
OSA-NO	6	35.3

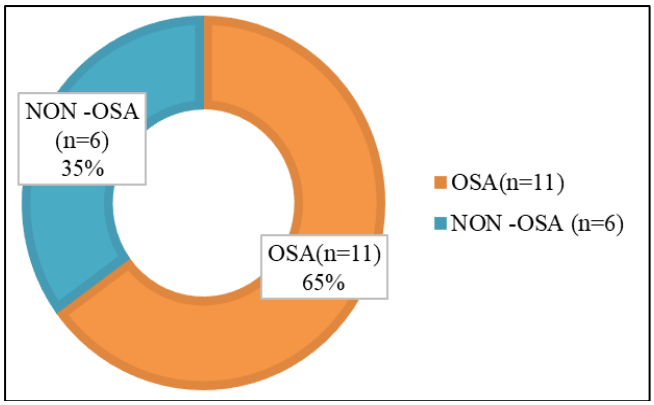

Fig4: Prevalence of PHTN in OSA patients with obesity.

Severities of PHTN in OSA patients were depicted in Table 5 and figure 5 using Pulmonary artery systolic pressure (PASP) in ECHO. In this severe PHTN were noted in higher proportion in OSA patients compared to NON OSA patients with OAD.

Table 5: Severity of Pulmonary HTN IN OSA and non OSA with OAD

Pulmonary HTN	OSA	NON OSA
<35 mmHg	3	1
35-50mmHg	4	0
50-60mmHg	4	0
>60mmHg	14	1

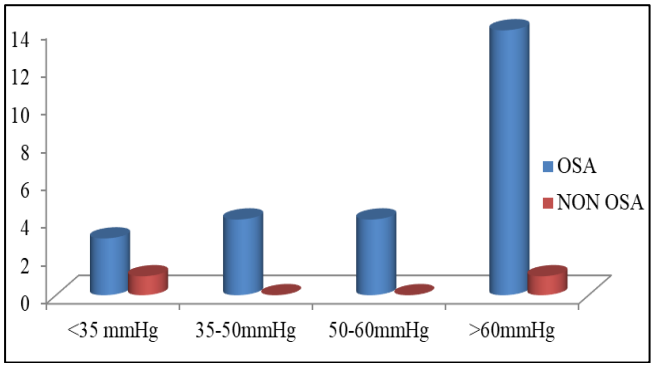

Fig 5: Severity of pulmonary HTN in OSA and non OSA with OAD

Table 6 and figure 6 showed severity of PHTN in obese patients.in this severe PHTN were observed more number in obese patients than non obese patients with OAD.

Table 6: Severity of PHTN in OSA with Obese and non-obese with OAD(BMI>25)

Pulmonary HTN In OSA	OBESE	NON OBESE
<35 mmHg	1	2
35-50mmHg	2	2
50-60mmHg	2	2
>60mmHg	8	6

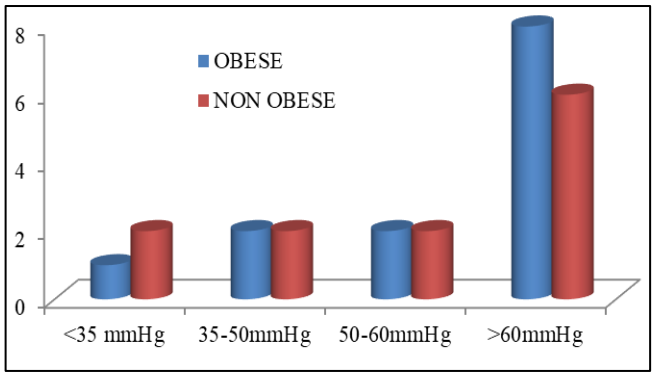

Fig 6: Severity of PHTN in OSA with obese and non-obese patients with OAD(BMI>25)

Conclusion

Pulmonary hypertension is invariably present in most of the OSA and Obese patients with obstructive airway diseases. Hence control of OSA by CPAP therapy will prevent or delay the development of pulmonary hypertension.

Reference

1. Guilleminault C, Tilkian A, Lehrman K, *et al*. Sleep apnea syndrome: state of sleep and autonomic dysfunction. J Neurol Neurosurg Psychiatry. 1977;40:718-25
2. Lopez AD, Shibuya K, Rao C, Mathers CD, Hansell AL, Held LS, *et al.* Chronic obstructive airway disease: Current burden and future projections. Eur Resp J. 2006;27:397-412
3. Barbé F, Pépin JL. Obstructive Sleep Apnoea. Eur Respir Society, 2015.
4. Amra B, Golshan M, Fietze I, Penzel T,Welte T, Correlation between chronic obstructive pulmonary disease and obstructive sleep apnea syndrome in a general population in Iran. Journal of Research in Medical Sciences, J Res Med Sci. 2011 Jul;16(7):885-889.
5. Bhavneesh Sharma *et al*., Obstructive Airway Disease and Obstructive Sleep Apnea: Effect of Pulmonary Function, Lung. 2011;189(1):37-41. doi:10.1007/s00408-010-9270-3.
6. Raj Kumar *et al*., Obstructive sleep apnoea and atopy among middle aged chronic obstructive pulmonary disease and bronchial asthma patients, J Assoc Physicians India. 2013 Sep;61(9):615-8.
7. Wan-Lu Sun *et al.*, Impact of obstructive sleep apnea on pulmonary hypertension in patients with chronic obstructive pulmonary disease, Chin Med J (Engl) 2019; Jun5;132(11):1272-1282.
8. Abou Shehata ME Mohamed E, El-DesokyAbd El-Razek Maaty Amina M, Abd-ElMaksoud Lucy A. Suliman, Pulmonary hypertension in obstructive sleep apnea hypopnea syndrome. Egyptian Journal of Chest Diseases and Tuberculosis. 2013;62(3):459-465.