EVALUATION OF NOCTURNAL OXYGEN DESATURATION IN COPD

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ABSTRACT

BACKGROUND

Patients of COPD become hypoxic during sleep to a significant extent. Florid hypoxic episodes occur during REM sleep secondary to central diminution in respiratory output, accentuated by hypotonia of postural muscles, intercostals and accessory muscles of respiration.

MATERIALS AND METHODS

This is a cross-sectional study carried out prospectively in Gayathri Vidya Parishad Institute of Healthcare and Medical Technology, Visakhapatnam, AP, India, to evaluate breathing disorders during sleep in COPD patients and to correlate them with the stage of the disease.

SAMPLE SIZE

A total of 36 COPD patients were enrolled into the study. They are classified into Mild, Moderate and Severe COPD categories in accordance to the Indian guidelines. The study was conducted between April 2014 and May 2016.

POLYSOMNOGRAPHY

Overnight sleep study was conducted using Compumedics Profusion Polysomnographic Machine. A total of 20 leads were utilised for the study. The sleep data recorded by the computer was manually scored for analysing Sleep stages, Apnoeas and Hypopnoeas. Sleep scoring was done according to R and K classification. Nocturnal oxygen desaturation is defined as >30% of total recording time with a SaO_2 <90% (or) nocturnal SaO_2 <85% for at least 5 minutes.

RESULTS

Out of the 36 patients enrolled into the study, 6 were having mild COPD, 22 had moderate COPD and 8 had severe COPD. Total number of patients who had significant oxygen desaturation during sleep were 5 (13.9%). Out of these, 1 patient (16.67%) belonged to Mild COPD, 1 (4.54%) belonged to Moderate COPD and 3 (37.5%) belonged to Severe COPD.

CONCLUSION

We conclude that in patients with COPD, daytime SpO_2 is the single most useful determinant that contributes to NOD; daytime hypercapnia being the other important factor. In Severe COPD group, daytime PaO_2 contributes to NOD whereas in Mild COPD, a raised AHI might explain the occurrence of NOD.

KEYWORDS

Nocturnal Oxygen Desaturation, REM Sleep, COPD.

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INTRODUCTION: All patients with COPD become more hypoxaemic during sleep than during restful wakefulness.[1-3] The drop in oxygen saturation during sleep is more than during exercise and because patients of COPD spend much more time in sleeping than exercising. Sleep is a more significant cause of hypoxaemic load for these patients.

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Nocturnal Oxygen Desaturation (NOD) occurs most markedly during Rapid Eye Movement (REM) sleep when florid hypoxaemia may occur.[4] During REM sleep, the marked reduction in ventilation secondary to central diminution in respiratory output occurs intermittently during periods of frequent eye movements[5] when tidal volume falls substantially. Also, there is hypotonia of postural muscles, the intercostals and accessory muscles of respiration[6] leading to decreased contribution of rib cage to ventilation.[7]

MATERIALS AND METHODS: The present study was a cross-sectional study prospectively carried out in the well-equipped sleep laboratory of Gayatri Vidya Parishad Institute of Healthcare and Medical Technology, Visakhapatnam, AP, India; with an aim to evaluate the

breathing disorders during sleep in patients with COPD and to correlate these disorders with the stage of the disease.

Study Period: The study Cohort was constituted by patients of COPD registered into Chest OPD or admitted in Indoor Units of Gayathri Vidya Parishad Institute of Healthcare and Medical Technology, Visakhapatnam, AP, from April 2014 to May 2016.

Sample Size: A total of thirty-six consecutive COPD patients who consented to be enrolled into the study were classified into Mild, Moderate and Severe stages based on the Indian Guidelines for the management of COPD, i.e. Mild COPD: FEV1/FVC <70%, FEV1 >80% predicted; Moderate COPD: FEV1/FVC <70%, FEV1 30-80% predicted; Severe COPD: FEV1/FVC <70%, FEV1 <30% predicted. The inclusion criteria followed for enrolling the patients in the study were Age >40 years; Clinical history consistent with COPD; Irreversible Airflow Obstruction, i.e. Forced Expiratory Volume in one second (FEV1)/Forced Vital Capacity (FVC) <70% and post-bronchodilator change in FEV1 <15% (or) if FEV1 <1.5 L change in FEV1 <200 mL.

Patients with active tuberculosis, congestive heart failure, chronic renal failure, morbid obesity (BMI >40), pregnant women, age <40 years and >80 years were excluded from the study.

METHODS: Detailed history, complete general and systemic physical examination and all relevant laboratory investigations were conducted as per the protocol.

Anthropometry: Weight and height were measured to the nearest 500 g and one cm, respectively and the Body Mass Index (BMI) was calculated based on the formula [BMI=weight (kg)/height^2 (m^2)]. Neck circumference (cm) was measured at the level of cricothyroid membrane.

Pulmonary Functions: Spirometric evaluation and bronchoreversibility testing was conducted in all the patients. The patients were instructed to withhold the morning dose of inhaled bronchodilators on the day of pulmonary function testing.

Arterial Blood Gas Analysis was done using ABL3 arterial blood gas analyser (Radiometer, Copenhagen). Based on the spirometric evaluation, patients were further classified into Mild, Moderate and Severe COPD groups in accordance to the Indian Guidelines for COPD. An informed consent was taken from each patient.

Polysomnography: Patients reported to the Sleep Laboratory at 8:00 pm on the day of their appointment. They were hooked to Compumedics Profusion Polysomnographic machine (Compumedics Private Limited 2001, USA) by standard gold cups/electrodes, after cleansing the area of attachment by spirit and Savlon. Thereafter, the patients were subjected to a full night sleep study (Overnight polysomnography). The electrode and sensor connection system utilises E-series EEG/PSG system in order to record the PSG study. The impedance of electrodes was checked and set to <10.

A total of 20 leads were utilised for the study. The various parameters monitored included Electroencephalogram (EEG), Electro-oculogram (EOG), Electrocardiogram (ECG), chin and leg electromyogram (EMG), nasal airflow, tracheal breath sounds, thoracic wall movements, abdominal movements, transcutaneous oxygen saturation and body position. The polysomnographic study was started at a time, which coincides with the normal sleeping habits of the patient. The sleep data recorded by the computer were manually scored for sleep stages, apnoeas and hypopneas. The sleep scoring was done according to R and K classification. Nocturnal oxygen desaturation is defined as >30% of total recording time with a SaO_2 <90% (or) nocturnal SaO_2 <85% for at least 5 minutes.

RESULTS: The mean age was 53.31 years (Range 40 to 77 years, 35 males, 1 female). The mean BMI was 18.424 (range 13.7 to 28.7), mean neck circumference was 374.903 (range 14 to 28). Most of the patients enrolled into the study were heavy smokers with the mean Pack - years of 30.69, (Range 15 to 60). Of these, 6 patients had mild COPD, 22 had moderate COPD and 8 had severe COPD. Mean age of mild, moderate and severe COPD patients are 58.17, 53.5 and 49.38, respectively.

	NOD (n=5)	non-NOD (n=31)	't' value	p value
AGE	52.6±5.59	53.48±8.85	1.25	p>0.05
BMI	18.42±2.09	18.43±3.49	0.02	p>0.05
NC	36.5±3.39	34.65±3.09	-7.17	p>0.05
FEV1/FVC	43.2±13.16	53.45±11.91	10.28	p<0.05
FEV1%	39.2±25.34	51.74±18.80	7.71	p<0.05
PCO2	47.82±11.88	41.27±6.30	-11.02	p>0.05
PO2	67.28±16.54	80.42±9.36	15.19	p<0.05
O2 Sat	91.52±5.78	95.50±1.80	18.51	p<0.05
RDI	3.52±1.09	1.66±1.92	-12.20	p>0.05

Table 1: NOD and Non-NOD - Characteristics (n=36)

Parameter	r value	p value
Neck Circumference	0.239	>0.05
BMI	0.386	>0.05
FEV1% pred.	0.75	>0.05
FEV1/FVC	0.486	> 0.05
$PaCO_2$	-0.957	<0.05
PaO_2	0.844	>0.05
SpO_2	0.986	<0.01
RDI	0.436	>0.05

Table 2: Correlations: Nocturnal Oxygen Desaturation (n=5) Correlation between Nocturnal SpO_2 and Other Parameters

	Mild (n=6)		Moderate (n=22)		Severe (n=8)	
	t value	p value	t value	p value	t value	p value
AGE	-0.27	>0.05	0.78	>0.05	-0.03	>0.05
BMI	0.03	>0.05	0.88	>0.05	-0.18	>0.05
NC	-0.30	>0.05	2.04	<0.05	0.57	>0.05
FEV1/FVC	-0.47	>0.05	-0.07	>0.05	-0.13	>0.05
FEV1%	-0.25	>0.05	-0.28	>0.05	-1.63	>0.05
PCO2	-0.38	>0.05	-0.27	>0.05	1.55	>0.05
PO2	0.75	>0.05	-1.15	>0.05	-4.24	<0.05
O2 Sat	0.78	>0.05	-0.71	>0.05	-2.14	<0.05
RDI	0.93	>0.05	0.59	>0.05	3.97	<0.05

Table 3: Nocturnal Oxygen Desaturation

Parameters	r value	p value
PaO_2	0.99	<0.05
SpO_2	0.74	>0.05
RDI	0.97	>0.05

Table 4: Correlation: NOD in Severe COPD (Correlation between Nocturnal SpO_2 and Other Parameters)

Pulmonary Functions: The mean value of FEV_1/FVC ratio was 52.03 (Range 26 to 79), mean FEV_1 % pred. was 50 (Range 19 to 83). The daytime Arterial Blood Gas (ABG) analysis showed that the Mean PH was 7.41 (Range 7.34 to 7.49), the mean PCO_2 was 42.18 (Range 32.3 to 68.3). Out of the 36 patients, 6 patients had daytime hypercapnia (PCO_2 >45 mmHg). The mean PO_2 was 79.156 (Range 49 to 107.2). Two patients had daytime hypoxia (PO_2 <60 mmHg). The mean Arterial Oxygen saturation was 95.033% (Range 82 to 98.2) with one patient having a SpO_2 <90%. Total number of patients who had significant oxygen desaturation during sleep were 5 (13.9%). Out of these, 1 patient (16.67%) belonged to Mild COPD, 1 (4.54%) belonged to Moderate COPD and 3 (37.5%) belonged to Severe COPD.

Analysing the characteristics of the NOD and NND in all the 3 groups of COPD combined revealed that the patients with NOD had significantly lower FEV_1/FVC, FEV_1%, PO_2 and SpO_2. (Table 1) Regression analysis revealed that awake SpO_2 had a highly significant positive correlation ($r=0.986$, $p<0.01$) and Daytime $PaCO_2$ had a significant negative correlation with nocturnal O_2 saturation ($r=-0.957$, $p<0.05$). Awake PaO_2 also showed to have a positive correlation though not to a significant extent.

No significant correlation is found to exist between nocturnal oxygen saturation (avg.) and the anthropometric variables, viz.; NC and BMI, or with the spirometric variables, viz.; FEV_1/FVC and FEV_1% (Table 2). In the subgroup analysis of COPD, unpaired 't' test was done to distinguish between NOD and NND in the respective groups. In mild COPD group, no significant difference is found to exist between NOD and NND; however, the patient had concomitant OSA (AHI >5). In Moderate COPD, the patient with NOD had significantly larger neck circumference. In Severe COPD, awake PaO_2, SpO_2 and RDI were significantly lower in NOD patients compared to NND in this group. (Table 3) Regression analysis in this group revealed that patients with NOD had a significant correlation between awake PaO_2 and nocturnal oxygen saturation (Avg.) (Table 4).

DISCUSSION: There has been a great deal of quest to know the characteristics of patients with COPD who might have nocturnal oxygen desaturation during sleep. Thomas VD et al documented that in their study on a south Indian COPD patients, 46.6% experienced NOD. They observed that desaturators had significantly lower awake oxygen saturation and PaO_2 when compared to non-desaturators and concluded that daytime SpO_2 is the only single

predictor of nocturnal oxygen desaturation in these patients.[8] Little et al studied daytime ABG and nocturnal SpO_2 in 33 patients with stable COPD. They found that the patients who experienced severe nocturnal desaturation had significantly lower mean PaO_2 and SpO_2 values as compared to those who did not have desaturation.[6]

We observed that nocturnal oxygen desaturators not only had significantly lower SaO_2 ($r=0.986$, $p<0.01$), but also had a higher $PaCO_2$ ($r=-0.957$, $p<0.05$) when compared to non-desaturators. The various definitions of NOD followed in the literature might be responsible for the increased occurrence of NOD in the above studies. We also observed that awake PaO_2 is found to have a weak correlation with NOD. Sharma SK et al observed that daytime PCO_2, FEV_1 had an indirect correlation with desaturation during sleep and concluded that BMI is the only factor responsible that can predict NOD in these patients.[9] Most of the patients enrolled in this study had respiratory failure and there was no mention of the total number of patients who had NOD. De Angelis et al reported that awake PCO_2 >50 mmHg is highly indicative of NOD; the other useful indicators were mentioned as a decreased FEV_1 (<49%) and increased BMI.[10] We did not observe any correlation between NOD and the anthropometric variables like neck circumference and BMI.

Subgroup analysis of COPD patients revealed that no significant correlation exists between Avg. minimum SpO_2 in mild COPD patients. In moderate COPD patients, Daytime PaO_2 ($r=0.609$, $p<0.01$) and Daytime SpO_2 ($r=0.623$, $p<0.01$) showed significant positive correlation with Avg. minimum nocturnal SpO_2. In patients with severe COPD, Daytime PaO_2 is found to have a highly significant correlation with Nocturnal O_2 saturation ($r=0.01$, $p<0.01$). Daytime SpO_2 also showed significant positive correlation ($r=0.734$, $p<0.05$) whereas Daytime $PaCO_2$ showed significant negative correlation with Nocturnal O_2 saturation ($r=-0.785$, $p<0.05$).

S. K. Sharma et al concluded that in their study, there was no significant correlation between any of the pulmonary function parameters and the parameters related to sleep disordered breathing such as AHI, apnoea, hypopnoea and episodes of nocturnal oxygen desaturation.[9] Similarly, we did not observe any significant correlation between awake spirometric variables viz.; FEV_1/FVC and FEV_1 % pred. and Nocturnal O_2 desaturation. Appelberg et al reported that FEV_1/FVC was slightly, but significantly lower in subjects who had nocturnal apnoeas and desaturation as compared to those who did not have these.[7] Sanders MH et al observed that FEV_1/FVC <65% independently predicted NOD in patients with mild COPD even after adjusting the awake SpO_2.[11]

We observed that the patient who had NOD in the Mild COPD group had concomitant Obstructive Sleep apnoea, which might be responsible for the florid desaturation during sleep in this group. Similarly, in Moderate COPD group, we observed that the patient with NOD had a significant increase in neck circumference when compared to those without NOD, which is an independent risk factor for OSA. In the group of Severe COPD, we found that awake PaO2 ($r=0.9977$, $p<0.05$) is the most useful factor that can predict NOD in this group of patients.

We conclude that in patients with COPD on the whole, daytime SpO_2 is the single most useful determinant that contributes to NOD, daytime hypercapnia being the other important factor. In Severe COPD group, daytime PaO_2 contributes to NOD whereas in Mild COPD a raised AHI might explain the occurrence of NOD.

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