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ORIGINAL ARTICLE

Pulmonary Hypertension in Recently Diagnosed Obstructive Sleep Apnea Patients

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a condition characterized by daytime sleepiness, with occurrence of ≥ 5 obstructive events per hour during sleep, associated with cyclic oxygen desaturation. The cumulative effect of intermittent hypoxia can lead to pulmonary hypertension (PH). The study aimed to assess the association of PH with recently diagnosed OSA.

Methods: Fifty newly diagnosed OSA patients with PH were included. Their PH was diagnosed based on echocardiographic findings. They seemed to have PH, if mean pulmonary artery pressure (mPAP) ≥25 mmHg.

Results: 42 patients (84%) reported daytime sleepiness, 41 (82%) were snorers, 37 patients (74%) experienced witnessed apnea, 36 patients (72%) reported tiredness or fatigue, 27 patients (54%) suffered from choking and 22 patients (44%) reported nocturia. Mean snoring index was 304.85 (±290.60) events/hour. On the other hand, 20% of moderate OSA patients had mild PH, 33.3% had moderate PH and 46.7% had severe PH. Finally, the percentages of mild, moderate, and severe PH in severe OSA patients were 12%, 28% and 60% respectively. There was a significant association between OSA severity and both approximated pulmonary artery systolic pressure (PASP) & approximated mPAP. Approximated PASP was significantly higher in severe OSA than mild and moderate OSA (75.3 vs 69.8 vs. 52.3 mmHg respectively, p =0.018). Approximated mPAP was significantly higher in patients with severe OSA than patients with mild and moderate OSA (47.93 vs. 44.6 vs. 33.9 mmHg respectively, p =0.016

Conclusions: PH and its degree are directly correlated to OSA and its severity.

Key words: Echocardiography; Pulmonary Hypertension; Obstructive Sleep Apnea.

INTRODUCTION

Obstructive sleep apnea (OSA) is a condition characterized by daytime sleepiness with occurrence of five or more obstructive events per hour during sleep (apneas and hypopneas)[1].

Most of OSA patients experience cyclical oxygen desaturations during sleep. These episodes occur several times an hour, where each last for a period between 10 to 40 seconds. They are followed by arousals and recovery of oxygen saturation. Yet, the cumulative effect of resulting intermittent hypoxia is both polycythemia and pulmonary hypertension (PH) [2,3].

The PH associated with OSA can be attributed to multiple pre-causes, examples of which are remodeling of pulmonary arterioles, hyperactivity to hypoxia, left atrial enlargement and left ventricular diastolic dysfunction [4].

Although PH occurs as a consequence of OSA, symptoms may not be present because of obesity and a lack of physical exertion. Symptoms of PH can also get overshadowed by those of other cardiovascular abnormalities, if present [5].

Recent studies have shown that PH was found in 20% to 40% of patients with OSA after excluding other known cardiopulmonary disorders [4]. Other studies found that PH prevalence among OSA was as low as 3%–7% prevalence among men and 2%–5% prevalence among women [6]. The study aimed to assess the association of PH in recently diagnosed OSA patients.

METHODS

This study was conducted at Sleep Disordered Breathing Unit, Chest Department, Zagazig University Hospitals during the period between October 2019 to April 2020. The study was approved by the Institutional Review Board-Zagazig University (IRB-ZU). Written informed consents were obtained from all patients. Fifty newly diagnosed OSA patients suffering from PH were included in our study, among whom 30 were males and 20 were females. Their ages range were from 47 to 66 years and the mean age was 57.80 ±

6.6years. The study was done according to The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

***Inclusion Criteria*:** All patients were newly diagnosed OSA patients, diagnosed by performing full night polysomnographic sleep study, whose echocardiographic findings elucidate presence of PH. These patients seemed to have PH if approximated mPAP ≥25 mmHg. [7]

***Exclusion Criteria*:** Patients aged ≤ 18 years, Patients having secondary PH (due to pulmonary diseases, or left ventricular heart diseases, or chronic thromboembolic disorders), Patients whom are on long term oxygen therapy (LTOT), Patients suffering from respiratory neuromuscular weaknesses or chest wall deformities, Patients that have end-organ failure and/or malignancies and patients complaining of obesity hypoventilation syndrome.

***Study Design: Clinical study* (**Cross-over design)

82 patients were recruited, where 12 patients refused to participate in the study. Of participated patients 8 had already been diagnosed as COPD and 5 patients were on long term oxygen therapy so they were accordingly excluded. We lost contact with 7 patients, during the follow up, so follow-up ECHO couldn't be performed. Finally, the number of remaining patients who were included in our study was 50 patients.

***Sample Size*:** The estimated sample was 46 patients (OPEN EPI was used.), as PAP in OSA was found to be 35±10 pre-and 26±11.5 Post treatment at 95% CI and 80% Power.

Methods: Medical history of the patients was reviewed especially, daytime and nighttime symptoms of OSA (e.g. chronic snoring, witnessed apneas, daytime hypersomnolence, nightmares, choking and nocturia) [8]. Comorbidities including, hypertension, diabetes mellitus, atrial fibrillation, stroke, hypothyroidism, renal and hepatic impairment were screened for and documented. Stop-Bang scale of ≥ 3 and ESS scale of ≥10 was used to help in the diagnosis and monitoring of the patients [9,10]. Both general and local chest examinations were undergone to all our patients. This included measuring neck circumference and body mass index (BMI) [11].

All our patients obtained chest x-ray (posterior-anterior and lateral views). Some had high resolution computed tomography (HRCT) or computed tomography pulmonary angiography (CTPA) done, this was done when needed to exclude underlying diseases e.g. connective tissue diseases or chronic thromboembolic pulmonary hypertension (CTEPH) respectively. Pulmonary functions tests were done using a portable spirometer (Spirotube PC Spirometer, Thor Laboratories, Budapest, Hungary), for excluding pulmonary causes of secondary PH. Similarily, Arterial blood gases analysis was valuable to exclude patients in critical condition.

Electrocardiogram (ECG) was done to find ECG abnormalities related to PH. These included P pulmonale, right axis deviation, RV hypertrophy, RV strain, right bundle branch block, and QTc (corrected Q-T interval) prolongation [11]. We thoroughly reviewed the polysomnography sleep study (PSG) reports of our patients focusing on apnea–hypopnea index (AHI), to assess OSA severity. (AHI<5) was considered as normal, (AHI 5–14) as mild, (AHI 15–29) as moderate, and (AHI≥30) as severe [8].

Oxygen desaturation index (ODI), which is the number of significant oxygen desaturations per sleep hour was recorded, as well as the maximum systolic blood pressure and the snoring index (SI), which is the number of snores per hour of sleep. A significant oxygen desaturation was scored, when SaO_2 decreased >4% below the immediately preceding baseline. The normal person's snoring index is up to 19 [12,13].

All enrolled patients were evaluated by trans-thoracic echocardiography using ultrasound system (Vivid I, GE Healthcare, Little Chalfont, UK) with a 2.5 MHz transducer. Certain measurements were then used to calculate mPAP. Patients were considered to have PH, if mPAP ≥25 mmHg and were classified into mild (25–40 mmHg), moderate (41–55 mmHg) and severe (>55 mmHg) degrees [14].

***Statistical Analysis*:** All data were collected, tabulated and statistically analyzed using SPSS 22.0 for windows (IBM Inc., Chicago, IL, USA), MedCalc 13 for windows (MedCalc Software bvba, Ostend, Belgium) and NCSS 12 Statistical Software (2018) (NCSS LLC., Kaysville, Utah, USA). Continuous Quantitative variables were expressed as the mean ± SD & median (range) and categorical qualitative variables were expressed as absolute frequencies (number) & relative frequencies (percentage). Continuous variables were checked for normality by using Shapiro-Wilk test. The Mann Whitney U test was used to compare two groups of non-normally distributed variables. Categorical data were compared using Chi-square test or Fisher's exact test, when appropriate. The Wilcoxon Signed Ranks test was used to compare between two dependent groups of non-normally distributed variables. The ANOVA test was used to compare between three groups of normally distributed variables. All tests were two sided. p-value < 0.05 was considered statistically significant (S), p-value < 0.001 was considered highly statistically

significant (HS) and p-value ≥ 0.05 was considered statistically insignificant (NS).

RESULTS

Our study included 60% males. The mean age of our patients was 57.80 years. 70% of our patients were non-smokers. Mean body mass index was 40.41 kg/m2. Diabetes Mellitus (DM) was the most prevalent co-morbidity (54%) followed by hypertension (44%). Mean Neck circumference was 42.70 cm. Mean ESS score was 13.06. Where 56% of our patients had stop BANG score ≥5 (Table 1).

Regarding frequency of sleep symptoms among the studied patients. The most prevalent symptom was daytime sleepiness (84% of our patients), while the least prevalent symptom was nocturia (44%). 82 % had snoring, 74 % had witnessed apnea, 72 % had tiredness or fatigue and 54% had choking. Mean duration of symptoms was 4.90 years and ranged from 3 to 10 years (Table 2).

Regarding the frequency of ECG Changes in all studied populations, the most prevalent changes were right axis deviation (96% of the patients) all of whom had P pulmonale. 64% had R/S ratio more than 1 in V1. 64% had R wave more than 7mm in V1. 78% had rSR complex in V1 with R>10mm (Table 3).

Our patients mean AHI (±SD) was 33.85 (± 1.67) events per hour(e/h) and ranged from 8.10 to 60.30 e/h. Their mean O2 desaturation index was 39.77 (± 20.93) e/h and ranged from 18.90 to 83 e/h. Mean minimum saturation was 85 (±3.61) % and ranged from 82 to 90 %. Mean snoring index was 304.85 (±290.60) e/h and ranged from 75 to 934 e/h (Table 4).

In the studied patients, severe OSA was the most prevalent (50 % of the patients), while mild and moderate grades were represented in 20% & 30 % respectively. Also 46% of the patients had severe PH, while mild and moderate forms were presented in 24 % & 30 % respectively (Table 5).

Table (6) shows that there was a significant association between OSA severity and PH severity, where 60% of mild OSA patients had mild PH, while 60% of severe OSA patients had severe PH (p-value=0.027).

Table (7) shows that there was a significant association between OSA severity and approximated PASP & mPAP. Approximated PASP was significantly higher in patients with severe OSA than patients with moderate and mild OSA (mean: 75.3 vs. 69.8 vs. 52.3 mmHg respectively, p-value=0.018). Likewise, mPAP was significantly higher in patients with severe OSA than patients with moderate and mild OSA (mean: 47.93 vs 44.6 vs 33.9 mmHg respectively, p-value=0.016).

Table (1): Demographic data of the studied patients

Demographic data	All studied patients (N=50)	
	Number	%
Sex		
Male	30	60%
Female	20	40%
Age (years)		
Mean ± SD	57.80 ± 6.64	
Median (Range)	59.50 (47 – 66)	
Smoking		
Non-smoker	35	70%
Smoker	5	10%
Ex-smoker	10	20%
BMI (kg/m^2)		
Mean ± SD	40.41 ± 7.12	
Median (Range)	40 (29 – 51)	
Co-morbidities		
Hypertension	22	44%
IHD	9	18%
DM	27	54%
Neck circumference (cm)		
Mean ± SD	42.70 ± 4.11	
Median (Range)	44.50 (35 – 47)	
ESS score		
Mean ± SD	13.06 ± 1.94	
Median (Range)	10.50 (10 – 15)	
Stop BANG score		
<3	4	8%
3-4	18	36%
≥5	28	56%

Table (2): Frequency of sleep symptoms among studied patients

Sleep symptoms	All studied patients (N=50)	
	Number	%
Daytime Sleepiness		
No	8	16%
Yes	42	84%
Tiredness/Fatigue		
No	14	28%
Yes	36	72%
Choking		
No	23	46%
Yes	27	54%
Witnessed apnea		
No	13	26%
Yes	37	74%
Snoring		
No	9	18%
Yes	41	82%
Nocturia		
No	28	56%
Yes	22	44%
Duration of symptoms (years)		
Mean ± SD	4.90 ± 1.05	
Median (Range)	4.50 (3 – 10)	

Table (3): Frequency of ECG changes in all studied populations (N=50)

ECG changes	Frequency	
	No.	(%)
Right axis deviation	48	(96 %)
R/S ratio > 1 in V1(tall R, deep S)	32	(64 %)
R wave > 7mm in V1	32	(64 %)
rSR complex in V1 with R >10 mm	39	(78 %)
P pulmonale*	48	(96 %)

* P wave amplitude >2.5 mm in inferior leads (II, III, AVF) or > 1.5mm in V1/V2

Table (4): Polysomnographic data of the studied patients

Polysomnographic Data	All studied patients (N=50)		
	Mean ± SD	Median	(Range)
AHI(e/h)	33.85±1.67	29.65	(8.10 – 60.30)
O_2 desaturation index (e/h)	39.77±20.93	33.75	(18.90 – 83)
Minimum saturation (%)	85±3.61	81	(82 – 90)
Snoring index (e/h)	304.85±290.60	210.70	(75 – 934)

Table (5): Frequency of OSA and PH severity among studied patients

	All studied patients (N=50)	
	Number	%
OSA severity		
Mild	10	20%
Moderate	15	30%
Severe	25	50%
Pulmonary hypertension severity		
Mild	12	24%

Moderate	15	30%
Severe	23	46%

Table (6): Relation between OSA and PH severity among studied patients (N=50)

	Mild OSA (n=10)	Moderate OSA (n=15)	Severe OSA (n=25)	Test‡	P value (Sig.)
Mild pulmonary hypertension (n=12)	6 (60 %)	3(20 %)	3(12 %)	10.97	0.027 (S)
Moderate pulmonary hypertension (n=15)	3(30 %)	5(33.3 %)	7(28 %)		
Severe pulmonary hypertension (n=23)	1(10 %)	7(46.7 %)	15(60 %)		

‡ Chi-square test. p< 0.05 is significant. Sig.: Significance.

Table (7): Relation between obstructive sleep apnea severity and Echocardiographic findings (N=50)

	Mild (OSA)	Moderate (OSA)	Severe (OSA)	test‡	P
	Mean ± SD Median (Range)	**Mean ± SD Median (Range)**	**Mean ± SD Median (Range)**	0.28	0.018
Approximated PASP (mmHg)	52.3±1.3	69.8±3.6	75.3±2.8		
	51 (33-53)	68 (45-75)	71 (71-80)		
Approximated mPAP (mmHg)	33.9±0.9	44.6±2.9	47.93±2.1	0.25	0.016
	33 (31-43)	42 (39-58)	46 (45-61)		

‡One way ANOVA. p< 0.05 is significant.

Figure (1): Relation between OSA and PH severity among studied patients (N=50)

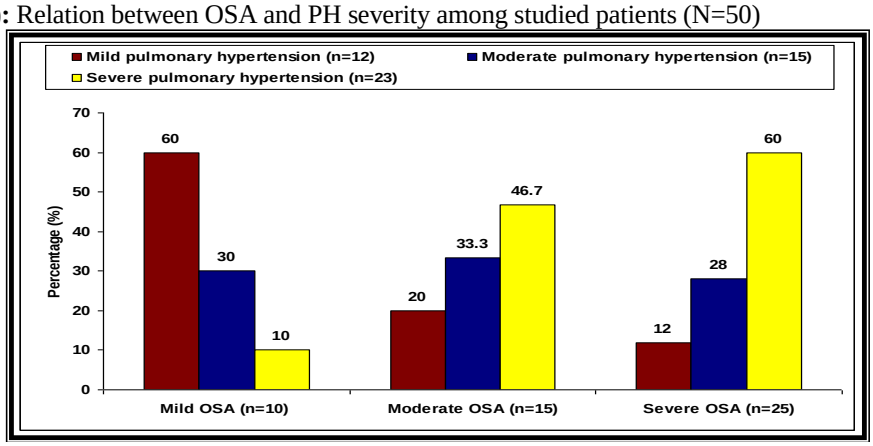
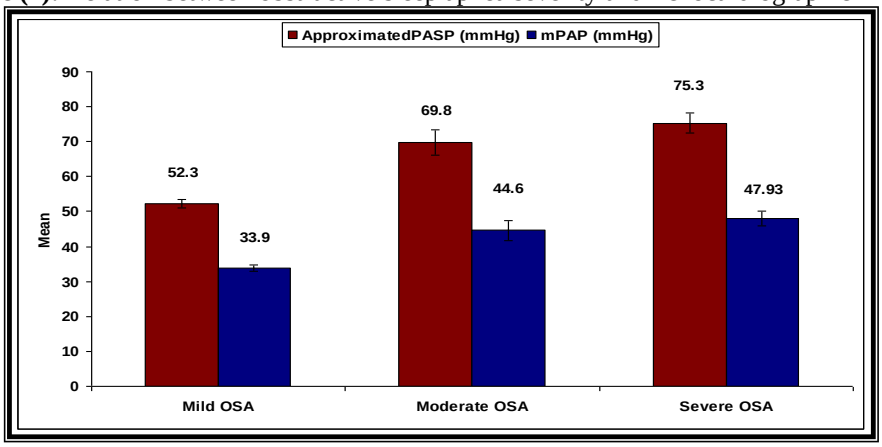


Figure (2): Relation between obstructive sleep apnea severity and Echocardiographic findings



DISCUSSION

This study aimed to study the association of PH with OSA. Such causal relationship can be interesting, especially with the increase in the OSA prevalence, that has reached around 1 billion people worldwide [15]. Our study included 50 patients, recently diagnosed with OSA with mean age of 57.80 ± 6.64 years, 60% of whom were

male patients and 70 % were non-smokers (Table 1). This data is quite similar to the results reported by many studies. Most of the patients included in these studies were non-smokers [16, 17].

The increase of OSA levels with age can be owed to age-related decrease in the size of the upper airway lumen in older people, especially in males. Structural changes to the upper airway's dimensions including, a lengthening of the pharyngeal airway and the descent of the hyoid bone, can lead to an increase in pharyngeal resistance. It is interesting that, even in healthy elderly people, pharyngeal resistance is increased compared with that in younger people, indicating a predisposition to airway collapse [18].

Regarding the associated comorbidities of the studied population, DM was the most prevalent (54%), followed by hypertension (44%) (Table 1). This can be attributable to the fact that most of our patients' OSA's severity is moderate to severe. This can be linked to the conclusion made by a multinational study, which found that the diagnosis and severity of OSA was associated with an increased likelihood of a concomitant diagnosis of type 2 DM [19]. Epidemiological evidence revealed a high prevalence of OSA in patients with type 2 DM. In a cross-sectional study, up to 23% of a diabetic population was found to have OSA [20]. Similarly in Mubaril et al., 36% of OSA patients had hypertension [21]. Another study revealed that OSA patients had significantly higher systolic blood pressure than controls [22].

Our patient's mean body mass index was 40.41 kg/m2 and their mean neck circumference was 42.70 cm (Table 1). This is in concordance with many reports that highlight the link between high BMI, neck circumference and occurrence of OSA. The high BMI of OSA patients in different studies indicates that obesity can be considered a main cause of OSA. This can be explained by the narrowing of the upper respiratory tract by the fat deposition around them. This is accompanied by decrease in muscle activity in this region, leading to apneic episodes, ultimately resulting in sleep apnea [23].

The mean neck circumference (NC) of Zayed et al study patients was 43.6 cm [24]. It is assumed that the increased deposition of fat or soft tissue in the region of the neck or adjacent to the upper airways is responsible for sleep apnea in obese individuals [25]. A large neck circumference probably reflects greater deposition of fat and soft tissue, which is more significantly associated with sleep apnea [25]. Meanwhile, another study endorsed the significant association between NC increases and associated with OSA diagnosis [26].

Regarding the screening questionnaires for sleep breathing disorders, our patients mean ESS score is 13.06±1.94, where 28 patients (56%) had stop BANG score ≥5 (Table 1). OSA patients usually have STOP-BANG score ≥ 3and ESS value ≥10 [27,28]. ESS score was more than that of OSA patients included in Guimarães, et al. study. Their mean ESS was found to be 11.8±5.3 cm [28]. In the same study the neck circumference of the OSA patients was very close to that of ours.

However, some recent studies have shown a decreased value of STOP BANG score in the diagnosis of OSA, while others suggested modifications to the way it is being calculated today. A recent study suggested simplifying STOPBANG to SOPBAG, while maintaining comparable screening performance and that it might be more practical to consider performing PSGs without the use of the STOPBANG [29]. Another study proposed the change of BMI value being used in the calculation of the score according to the race of the patient [30].

Regarding the frequency of sleep symptoms among studied patients, 42 patients (84%) reported daytime sleepiness, 41 (82%) were snorers, 37 patients (74%) experienced witnessed apnea, 36 patients (72%) reported tiredness or fatigue, 27 patients (54%) suffered from choking and 22 patients (44%) reported nocturia. Mean duration of symptoms was 4.90 years and ranged from 3 to 10 years (Table 2). The incidence and prevalence of reported symptoms is very variable among studies especially that the judgment is quite subjective and also because of the variability of AHI among different patients included in different studies.

In Nigro et al. study the most prevalent symptom was found to be snoring (in about 81.6% of the patients) [31]. Another study published in the same year showed that day time sleepiness is the commonest complaint [32].

In the current study, the most prevalent ECG changes among our patients were right axis deviation in 96% of the patients, all of whom had P-pulmonale. While 64% had R/S ratio more than 1 in V1 and 64% had R wave more than 7mm in V1, 78% had rSR complex in V1 with R>10mm (Table 3). In Shankar et al. study, the most common ECG abnormality among the patients was RS pattern with Deep S in Leads I and AVF [33].

Right ventricular hypertrophy (RVH) is an abnormal enlargement or pathologic increase in muscle mass of the right ventricle in response to pressure overload, most commonly due to severe lung disease [34]. Both the right ventricle's size and function are adversely affected by PH,

secondary to OSA. Right ventricular hypertrophy is usually associated with right axis deviation (axis greater than 90 to 100 degrees). Right atrial overload and ST-segment and T-wave abnormalities in the right precordial leads (used to be named "RV strain") are also usually present. They indicate sub-endocardial ischemia or right ventricular myocardium repolarization abnormalities. The right precordial leads (V1 and V2)'s tall R waves and left precordial leads (V5 and V6)'s deep S waves found in patients with pulmonary hypertension are caused by increase in the right ventricular forces [34].

Right ventricular hypertrophy can be suspected by the R: S ratio in V1 greater than 1, caused by the increase in the amplitude of the R wave and decrease in the depth of the S wave. Increased R: S ratio in adults occurs in an array of conditions including, right bundle branch block, posterior wall myocardial infarction, Wolff-Parkinson-White pattern, hypertrophic cardiomyopathy (septal hypertrophy), or even normal or positional variant [34].

ECG might have a prognostic role in patients with PH. So far, many studies have reported that some ECG patterns are commonly seen in PH. These patterns include, right atrial abnormalities, right axis deviation and right ventricular hypertrophy with strain pattern [35]. In a study, advanced pulmonary arterial hypertension was linked to the presence of qR in V1, where this sign was also shown to have a significant prognostic value [36]. In another study, the ECG of 23% of patients with PH was found to show right axis deviation (RAD) of QRS complex [37]. As far as we know not much has been reported about the ECG changes caused by PH secondary to OSA. We do hope that our data shall be useful for further research on this point.

Regarding the PSG parameters in the current study; mean (±SD) AHI was 33.85 ± 1.67 e/h and that of Oxygen Desaturation Index (ODI) was 39.77 ± 20.93. ODI can be used in both diagnosis and severity assessment of OSA (Table 4). ODI has a high reproducibility in the clinical setting. Faibus et al. concluded that ODI < 5 predicts an AHI < 5 with high sensitivity and specificity when measured simultaneously using the same oximeter during PSM recording [38]. The mean oxygen Nadir (mean minimum oxygen saturation) in our study was 85% (±3.61%) and ranged from 82 % to 90%. Mean snoring index was 304.85 (±290.60) e/h and ranged from 75 to 934 (Table 4). Snoring index is very useful as an indicator of OSA severity as shown by Maimoon et al study that found that snoring intensity was positively correlated not only with OSA severity, but also with neck circumference and BMI [39].

Considering the severity of our studied patients, severe OSA was more prevalent (50%), while mild and moderate grades were represented in 20%, 30 % respectively (table 5). Different results were found by Iannella et al. study, where 36.8 % of the OSA patients aged< 65 were mild, 30.5% were moderate and 32.6% were severe [40]. As regards to the severity of the PH 46% had severe PH, while mild and moderate forms were presented in 24% & 30 % respectively (table 5).

In our study, a significant association was found between OSA severity and PH severity. In the mild OSA patients, 60% had mild PH, 30 % had moderate PH and 10% had severe PH. On the other hand, 20% of moderate OSA patients had mild PH, 33.3% had moderate PH and 46.7% had severe PH. Finally, the percentages of mild, moderate and severe PH in our severe OSA patients were 12%, 28% and 60% respectively (Table 6).

There was a significant association between OSA severity and both approximated PASP & approximated mPAP. Approximated PASP was significantly higher in patients with severe OSA than patients with mild and moderate OSA. Approximated mPAP was significantly higher in patients with severe OSA than patients with mild and moderate OSA (Table7). This can be explained by the cyclical oxygen desaturations during sleep that occur in most OSA patients. The cumulative effect of intermittent hypoxia can lead to polycythaemia and PH. Both hypercapnia and nocturnal episodes of hypoxia can trigger pulmonary arteriolar constriction leading to acute reversible elevation in pulmonary artery pressures. Signaling pathways implicated in hypoxic vasoconstriction in PH include nitric oxide, endothelin, angiopoietin-1, serotonin, and NADPH-oxidase [5].

CONCLUSIONS

PH and its degree are directly correlated to OSA and its severity.

Conflict of interest: none

Financial disclosures: none

The authors alone are responsible for the content and writing of the paper.

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