The relationship between echocardiographic pulmonary steam velocity, respiratory functional tests and nocturnal oxygen desaturation in COPD patients

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Abstract: The aim of the study is to investigate the relationships between nocturnal oxygen desaturation levels and echocardiographic findings to diagnose earlier the pulmonary arterial hypertension (PH) in chronic obstructive pulmonary disease (COPD) and without daytime hypoxemia. We studied 55 consecutive patients with COPD (62.67 ± 8.20 years, 5 females) admitting our outpatient clinic and having consented to participate. All patients underwent routine biochemical, hematologic, thyroid function tests and chest x-ray. Spirometric tests, carbonmomoxide diffusion capacities, nocturnal sleep oxygen saturation level measures and 6 minute walking tests were performed to all patients. Systolic pulmonary steam velocity with pulsed–waved doppler and pulmonary artery pressures (PAP) of the patients were measured by transthoracic echocardiography. Thirty of the patients were found to have a normal PAP and 25 of them were found to have a high levels. A significant difference was determined between two groups in the terms of $Pa0_2$ ($p=0.030$), DLCO ($p=0.009$), percent sleep time spent with SaO_2 <90% (TST_{90}), ($p<0.0001$), the thickness of right ventricle ($p<0.0001$), minimum $Sa0_2$ levels in sleep ($p=0.0009$), mean $Sa0_2$ levels ($p<0.0001$) and the frequencies of nocturnal desaturations ($p=0.030$). PH was detected more frequently (11/12,:92%) among COPD patients with longer TST_{90} levels than 20% of total sleep time. As a conclusion, PH is a common clinical entity particularly among the patients having more frequent and longer nocturnal desaturation episodes. Observations of nocturnal oxygen desaturation on COPD patients may be a diagnostic tool to anticipate PH and to influence survivals.

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1. Introduction

Chronic obstructive pulmonary disease (COPD) is a highly prevalent, preventable and treatable disorder of the lung parenchyma and airways, characterized by persistent and progressive airflow limitation associated with hyperresponsiveness to noxious gases and particles. These patients had an unusual pattern of cardiopulmonary abnormalities with airway obstruction, severe hypoxemia, and a very low diffusing capacity for carbon monoxide. Parenchymal tissue destruction induced by inflammatory responses lead to breathlessness and hypoxia related symptoms (GOLD, 2011). The prevalence and burden of COPD are increasing due to continous exposure to risk factors and changing age structure of the population (Lopez et al., 2006; Mathers and Loncar, 2006). A new projection estimated COPD will be the third reason of death in 2020 (Mathers and Loncar, 2006). The direct and indirect costs of COPD may a represent a serious threat to economies worldwide.

The respiratory diseases such as COPD and sleep apnea are most frequently associated with pulmonary hypertension (PH) secondary to hypoxemia and categorized as WHO Group III PH (McLaughlin et al., 2009). Pulmonary hypertension detected echocardiographically in the vast majority of COPD patients (Scharf SM et al., 2002). Sleep disordered breathing can also produce PH especially when accompanied by severe hypoxemia. Treatment should be directed at the underlying disease including bronchodilator, inhaler corticosteroids, oxygen therapy and non-invasive mechanical ventilation (Scharf SM et al., 20024, McLaughlin et al., 2009).

In this study, we aimed to examine the relationship between echocardiographic screening of pulmonary hypertension, respiratory function tests and nocturnal oxygen desaturation (NOD) in COPD patients.

2.Materials and Methods

Patients and study design:

This investigation was an observational and descriptive type study conforming to the principles outlined by the Declaration of Helsinki and performed in pulmonary disease clinic of Istanbul University, School of Medicine. The study group consisted of 55 patients (62,67 ± 8,20 years, 5 females) having received a COPD diagnosis, having forced expiratory volume in 1 second/forced vital capacity (FEV_1/FVC) <70% and PaO2 ≥60 mmHg in resting room air. The main criteria for exclusion included ongoing or recent (in the past three months) exacerbation in COPD (worsening of

pulmonary symptoms leading to antibiotic and/or oral steroid treatment and/or hospitalization or an emergency visit). Patients with pulmonary arterial hypertension related with idiopathic, familial, connective tissue disease-associated, portopulmonary, congenital disease, HIV, and diet-drug related disease those were classified as WHO Group 1 PH (McLaughlin et al., 2009), venous thromboembolism and cardiac disease including diastolic dysfunction, LV dysfunction, significant valvular disease and the patients with symptoms and signs of obstructive sleep apnea were excluded from the study. The patients consented to participate to this study underwent physical examination, routine biochemical, hematologic, thyroid function tests, chest x-ray, diffusion capacity, spirometric tests including static and dynamic lung volumes, arterial blood gas analysis, nocturnal oxygen saturation measurements, 6 min walk test (6MWT) and transthoracic echocardiography (TTE) to identify the specific associated conditions.

Arterial blood gas analysis:

We measured the arterial blood parameters by using blood gas analyzer system (ABL 5, Radiometer AS, Copenhagen, Denmark) at room temperature after resting 15 minutes, 2 cc arterial blood samples were taken from the radial or the brachial arteries and were tested in the first 15 minutes.

Static and dynamic lung volumes:

Pulmonary functions including FEV_1, FVC and FEV_1/FVC were measured by using V-max 229 spirometry test equipment (Sensormedics, The cardio pulmonary care company, USA) in our clinic. At least 3 repeat measurements were done for each test (for less than 10% variability between results). The highest test value was considered as the test result. Residual volume (RV) and total lung capacity (TLC) were measured by gas dilution technique (nitrogen wash-out). The rates of the data according to predicted results were calculated by a software programme (Knudson RJ, et al., 1983).

Carbonmonoxide Diffusion Capacity:

Carbon monoxide diffusion capacity (DLCO) and DLCO per alveolar ventilation (DLCO/VA) of the patients was measured by a single breath technique using carbonmonoxide gas and V-max 229 spirometry. Reference values, calculations and the comparisons were calculated by a software programme (Burrows B et al., 1961).

Oxygen saturation measurements:

Nocturnal oxygen desaturation is typically used as desaturation greater than 3% for at least 5 minutes during sleep period in this study. Nocturnal saturation levels of the patients were recorded during a night period in our clinic by a pulse oximetry placed on finger. Basal oxygen saturation levels (SaO_2%), the mean time of nocturnal oxygen saturation, minimum and maximum oxygen saturation levels (min and max SaO_2%), percent sleep time spent with SaO_2 <90% (TST_{90}) were measured during sleep period by Sleepsound device (BEA, SA IEC 601-1 cl IIB, Belgium).

Six min walk test:

Pre and post 6MWT levels of SaO_2 and pulse rates were measured by pulse oxymeter device (BCI 3303, BCI International, Wisconsin USA) and recorded. At least 4% decrease in SaO_2 level was accepted as positive 6MWT after the physical effort (Little SA, et al., 1999).

Transthoracic echocardiography:

TTE is performed with HP. Sonor 2500 echocardiography equipment with high resolution to make the measurement and the echo reviewers were blinded to outcomes. All patients underwent standard TTE views including parasternal long axis, short axis, apical four chamber views as well as subcostal four chamber and short axis views. The studies were performed while the patient lying in supine and left lateral decubitus positions during quiet respiration. Right ventricular thickness measurement is made in apical four chamber view at the apex. Pulmonary arterial systolic steam was measured from mostly at parasternal short axis and subcostal long axis. Pulmonary systolic steam registrations were taken by pulsed-waved doppler. Pulmonary steam accelaration time (PSAT) was measured by using the time interval between systolic pulmonary steam sample and peak level of systolic pulmonary steam. PAP was calculated with a formula using the PSAT values (PAP=78- 0.52 x PSAT) and increased PAP >25 mmHg or PSAT ≥100 ms at rest were accepted as PH.

Statistical Analysis:

The statistical analysis was made with the use of a commercially available statistical package SPSS for Windows, version 15.0. Data were expressed as "mean (standard deviation; SD)", and percent (%) where appropriate. Descriptive statistics were provided for patient profile. ANOVA test was used for analysis of data showed normal distribution. The Mann–Whitney test was used to compare nonparametric two groups. Kruskal-Wallis test was used to compare more than two groups when the parameters did not distribute normally. P values <0.05 were considered statistically significant.

3. Results

The nocturnal saturation levels and echocardiographic pulsed-waved doppler results were analized. Demographical features and database of the patients were shown in **table-1**. The study population was divided into two groups according to PAP measured by echocardiographic PSAT. Group-I

included 25 patients (65,4±7,23 years) with PAP >25 mmHg and group-II included 30 patients (60,4±8,37 years) with PAP ≤25 mmHg (**table 2**). In the terms of $Pa0_2$ (mmHg), DLCO (%), hematocrit (%), percent sleep time spent with SaO_2 <90% (TST_{90}), right ventricular wall thickness (mm), minimum and mean $Sa0_2$% levels were significantly different between these two groups. On the other hand, $PaC0_2$ (mmHg), FVC (% of predict), FEV_1 (% of predict), DLCO/VA (%), TLC (% of predict), RV (% of predict), RV/TLC (%), pH, and daytime mean SaO_2% were not different. Twenty patients of the study group (20/55, 36%) had NOD less than 4% of total sleep time and PH was detected in only one of (1/20, 5%). PH was detected in 11 (92 %) of 12 cases with >20% of sleep time spent with SaO_2 <90% (TST_{90}). Six min walk test results were analyzed according to respiratory function tests and blood oxygen levels (**table-3**). Six min walk test positive group included 26 patients (63,96±9,72 years) and 6MWT negative group included 29 patients (61,52±6,51 years). PaO_2, FEV_1/FVC and daytime SaO_2 values were different between two groups. Arterial oxygen desaturation was observed in 8 of 15 (53%) patients with PaO_2 <65 mmHg and 4 of 17 (23%) patients with PaO_2 >75 mmHg in 6MWT. Only the one patient among 8 patients with normal PaO_2 levels (≥80 mmHg) showed desaturation in 6MWT. So, it is suggested that blood oxygen level may be a determinant of 6MWT. Forced expiratory volume in 1 second (%) value and NOD showed significantly negative association in this study (p=0.015). **Figure-1** shows the positive correlation between pulmonary arterial pressures and sleep time spent with SaO_2 <90%. PAP increased by the frequency of time spent with SaO_2 <90%. The relationsips between mean SaO_2 (%) values, hematocrit (%) levels and PAP (mmHg) were shown in **Figure-2.**

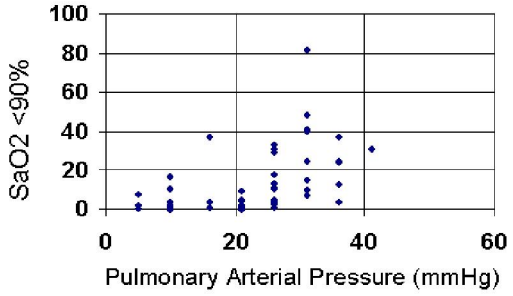

Figure-1: The relation between pulmonary arterial pressure and percents of total sleep time spent with SaO2 <90%

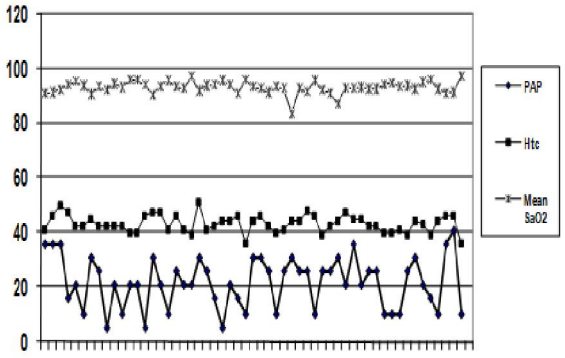

Figure-2: The relationsips between mean arterial oxygen saturation, hematocrit and pulmonary arterial pressure (PAP) levels.

Table-1: General charactheristics and spirometric results with blood gas analysis of the study group

Charactheristics	**n**	**Mean ± SD (Minumum-Maximum) values**
Age (years)	55	62.67 ± 8.20 (38-78)
Smoking (pack-year)	55	47.73 ± 25.22 (10-120)
BMI (kg/m^2)	55	27 ± 4.53 (17-40)
TTSF (mm)	55	10.84 ± 5.61 (3-26)
Htc (%)	55	43.40 ± 3.13 (36-51)
$Pa0_2$ (mmHg)	55	71.25 ± 7.68 (60-94)
$PaC0_2$ (mmHg)	55	42.27 ± 4.68 (33-54)
FVC (% of predict)	55	75.02 ± 16.95 (37-107)
FEV_1 (% of predict)	55	44.58 ± 12.93 (19-70)
TLC (% of predict)	44	130.32 ± 47.54 (74-326)
DLCO (% of predict)	52	67.51 ± 22.22 (19-124)
DLCO/VA (%)	52	68.43 ± 19.30 (26-111)
$Sa0_2$ (%)	55	93.80 ± 1.76 (90-97)

SD: standart deviation, **BMI:** body mass index, **TTSF:** thickness of triceps skin fold, **Hct:** hematocrit, **$Pa0_2$**: Partial arterial oxygen saturation, **$PaC0_2$:** Partial arterial carbondioxide saturation, **FVC:** forced vital capacity, **FEV_1:** forced expiratory volume in one seconds, **TLC:** total lung capacity, **DLCO:** carbonmonoxide diffusion test, **DLCO/VA:** volume adjusted carbonmonoxide diffusion test, **$Sa0_2$**: arterial oxygen saturation.

Table-2: Comparison of the results between including group-I (PAB<25 mmHg) and group-II (PAB>25 mmHg) echocardiographic pulmonary arterial pressure

Parameters	Non PH group (PAP <26mmHg)		PH group (PAP ≥26mmHg)		p
	n	mean value ± SD	n	mean value ± SD	
PaO_2 (mmHg)	30	73.47±7.83	25	68.60±6.71	**0.030***
$PaCO_2$ (mmHg)	30	42.57±5.10	25	41.92±4.19	0.641
FVC (% of predict)	30	75.70±16.57	25	74.20±17.71	0.630
FEV_1 (% of predict)	30	45.63±11.63	25	43.32±14.48	0.417
DLCO (% of predict)	28	75.75±18.34	25	58.28±22.88	**0.009***
DLCO/VA (%)	28	70.96±18.48	25	65.60±20.17	0.465
TLC (% of predict)	25	124.56±30.84	19	137.89±63.44	0.974
RV (% of predict)	25	197.84±74.63	19	238.50±150.78	0.678
RV/TLC (%)	25	57.91±9.98	19	60.60±13.65	0.654
TST_{90} (%)	30	4.74±7.37	25	22.07±18.60	**< 0.0001***
RVD (mm)	30	24.20±1.73	25	23.36±2.84	0.274
TRVW (mm)	30	4.31±0.54	25	5.24±0.98	**<0.0001***
Max 0_2 (%)	30	98.73±0.94	25	98.16±1.49	0.258
Min 0_2 (%)	30	81.27±8.39	25	74.88±11.33	**0.0009***
$Sa0_2$ (%)	30	94.25±1.48	25	91.60±2.17	**<0.0001***
NOD frequency	30	35.23±28.83	25	55.12±38.06	**0.030***
Test time (hours)	30	6.54±1.41	25	6.86±0.98	0.465
pH	30	7.39±2.82	25	7.39±2.61	0.654
Daytime $Sa0_2$ (%)	30	94.30±1.34	25	93.20±2.02	0.51

*: statistically significant, **SD**: standart deviation, **PH:** pulmonary hypertension, **PAP:** mean pulmonary arterial pressure, **$Pa0_2$**: Partial arterial oxygen saturation, **$PaC0_2$**: Partial arterial carbondioxide saturation, **FVC:** forced vital capacity, **FEV_1**: forced expiratory volume in one seconds, **DLCO**: carbonmonoxide diffusion test, **DLCO/VA**: volume adjusted carbonmonoxide diffusion test, **RV:** residuel volume of the lung, **TLC**: total lung capacity, **$Sa0_2$**: arterial oxygen saturation, **TST_{90}:** total sleep time spent with SaO2 <90% (% of total sleep time), **RVD**: Right ventricular diameter, **TRVW**: thickness of the right ventricle wall, **NOD frequency**: The frequency of nocturnal desaturation, **Min and Max 0_2:** minimum and maxiumum oxygen saturation levels during sleep period. **Daytime SaO_2 %:** The mean value of oxygen saturation (%) in day time.

Table 3: Comparison of the parameters (the mean values ± SD) between the patients with positive 6MWT and the patients with negative 6MWT

Parameters	Six minute walking test positive (n=26)	Six minute walking test negative (n=29)	p
Age (year)	63.96 ± 9.72	61.52 ± 6.51	0.188
PaO_2 (mmHg)	69.12 ± 7.42	73.17 ± 7.52	0.031*
$PaCO_2$ (mmHg)	41.58 ± 4.70	42.90 ± 4.60	0.217
FVC (% of predict)	72.19 ± 20.64	77.55 ± 12.65	0.221
FEV_1 (% of predict)	39.88 ± 13.02	48.79 ± 11.50	0.015*
FEV_1/FVC (%)	44.77 ± 12.56	50.79 ± 10.69	0.036*
DLCO (% of predict)	63.04 ± 24.58	72.78 ± 18.64	0.059
DLCO/VA (%)	68.08 ± 20.70	68.78 ± 18.23	0.922
TLC (% of predict)	128.95 ± 52.36	131.57 ± 43.84	0.589
RV (% of predict)	218.38 ± 127.61	212.70 ± 103.30	0.860
PAP (mmHg)	23.81± 9.53	19.69 ± 9.31	0.118
<90 SaO_2 (%)	13.18 ± 13.46	12.11 ± 18.00	0.183
MSS	92.99 ± 1.61	93.10 ± 2.72	0.332
Daytime SaO_2 %	93.23 ± 1.84	94.31 ± 1.54	0.021*

*: statistically significant, **SD:** standart deviation, **$Pa0_2$:** Partial arterial oxygen saturation, **$PaC0_2$**: Partial arterial carbondioxide saturation, **FVC:** forced vital capacity, **FEV_1:** forced expiratory volume in one seconds, **DLCO:** carbonmonoxide diffusion test, **DLCO/VA**: volume adjusted carbonmonoxide diffusion test, **TLC**: total lung capacity, **$Sa0_2$**: arterial oxygen saturation, **<90 SaO_2 (%):** the rate of desaturated time to all sleep time, **RV**: residual volume of the lung, **PAP**: Mean pulmonary arterial pressure, **RVD**: Right ventricular diameter, **MSS:** mean saturation values during sleep, **Daytime SaO_2 %:** The mean value of oxygen saturation (%) in day time.

4.Discussion

The cardiovascular consequences of COPD associated comorbidities especially pulmonary hypertension (PH) have been of significant interest in recent years. PH progresses rapidly to right heart failure and death and naturally it is a poor prognostic factor in COPD. The term PH refers to the presence of abnormally high pulmonary vascular pressure. Right heart catheterisation is the gold standard for the diagnosis of PH. The conventional definition of PAH used in clinical studies includes an mPAP of greater than 25 mm Hg at rest in the setting of a normal pulmonary arterial wedge pressure of 15 mm Hg or less with a PVR greater than 3 Wood units (Simonneau G et al., 2009; Farber HW et al., 2004). The use of echocardiography in the setting of suspected pulmonary hypertension is to be used as a screening test per ESC and AHA guidelines because it is non-invasive, widely available and relatively inexpensive. The correlation coefficient between systolic PAP estimated from echocardiography versus measured by right heart catheterisation was 0.70 (95% CI 0.67 to 0.73). The sensitivity and specificity for echocardiography for diagnosing PH was 83% and 72% respectively. The diagnostic OR was 13 (95% CI 5 to 31) (Janda S et al., 2011). Swanson et al.,(2008) analyzed echocardiography and right heart catheterization in PH. Right heart catheterization demonstrated PH in 75% while no PH was confirmed in 12%. Correlations existed between right ventricular systolic pressure with pulmonary artery systolic pressure ($r=0.66$; $p<0.0001$) and mean PAP ($r=0.69$; $p<0.0001$). Fisher et al., (2007) in patients with emphysema being evaluated for lung volume reduction surgery, Doppler echocardiography has been used to screen for PH as an indicator of increased peri-operative risk. To determine the accuracy of this test, the present authors compared the results of right heart catheterisations and Doppler echocardiograms in 163 patients participating in the cardiovascular substudy of the National Emphysema Treatment Trial. Substudy patients had both catheterisation and Doppler echocardiography performed before and after randomisation. In 74 paired catheterisations and echocardiograms carried out on 63 patients, the mean values of invasively measured pulmonary artery systolic pressures and the estimated right ventricular systolic pressures were similar. However, using the World Health Organization's definitions of PH, echocardiography had a sensitivity of 60%, specificity of 74%, positive predictive value of 68% and a negative predictive value of 67% compared with the invasive measurement.

Right atrium and right ventricle enlargement, reduced right ventricle function, displacement of the intraventricular septum, tricuspid regurgitation, the Tei index, and pericardial effusion have been evaluated by echocardiography (McLaughlin et al., 2009). Per 2009 ESC guidelines, PH is likely with a tricuspid regurgitation velocity > 3.4 m/s. Another practical echocardiographic method is estimating right ventricular systolic pressure by using the modified Bernoulli equation (Fisher MR et al., 2007; Swanson KL et al., 2008). In the absence of other potential etiologies of PH, such as left heart disease or advanced lung disease, an estimated right ventricle systolic pressure of greater than 40 mmHg, right atrium or right ventricle enlargement or intraventricular septal flattening generally warrants further evaluation in the patient with unexplained dyspnea. ACCF/AHA 2009 Expert Consensus Document state that "all patients that are still suspected of having PAH after noninvasive evaluation should undergo RHC (right heart catheterization) prior to initiation of therapy" (McLaughlin VV et al., 2009). In this study, PH was screened by pulsed waved Doppler echocardiographic PSAT measurements and we accepted the values greater than 100 ms or PAP >25 mmHg at rest as PH.

Gas exchange abnormalities of the lungs result in hypoxemia, hypercapnia and alveolar hypoventilations, reduces pulmonary vascular bed further worsen the ventilation/perfusion abnormalities (Rodriguez-Roisin R et al., 2009). There is an inflammatory response in vessels similar to that seen in the airways and evidence of endothelial dysfunction (Peinado VI et al., 2008). Pulmonary hypertension may develop late in the course of COPD and is due mainly to hypoxic vasoconstriction of small pulmonary arteries, eventually resulting in structural changes that include intimal hyperplasia and later smooth muscle hypertrophy/hyperplasia (Peinado VI et al., 2008). The loss of the pulmonary capillary bed in emphysema may also contribute to increased pressure in the pulmonary circulation.

The most important etiological factor for development of PH in patients with COPD is exposure to severe hypoxia ($PaO2 <60$ mmHg) in long durations. Activation of carotid chemoreceptors by hypoxemia triggers arteriolar vasoconstriction and systemic catecholamine secretion. This response is most marked in the systemic vascular bed at oxyhemoglobin saturation levels lower than 65% and leads to transient hypertension (Marrone O et al., 2006). Patients with COPD may show slow, progressive deteriorations in arterial blood gases during the night, particularly during rapid eye movement (REM) sleep. This is mainly due to hypoventilation, while a deterioration of ventilation/perfusion mismatch plays a minor role. The severity of gas exchanges alterations is proportional to the degree of impairment of diurnal pulmonary function tests, particularly of PaO_2 and of $PaCO_2$ in arterial blood (Marrone O et al., 2006).

Oximetry is easy to perform and to tolerate

and preferrable to monitorize SaO_2 during sleep period in most COPD patients, and inexpensive according to polysomnography test. The mean and the lowest nocturnal SaO_2 levels, and TST_{90} levels are measured. The measurement of the mean SaO_2 level is quite reproducible. TST_{90} is probably the most generally considered SaO_2 parameter, as it is used to distinguish patients with mild diurnal hypoxia into desaturators and nondesaturators (Levi-Valensi P et al., 1990); however, it is highly variable between nights, suggesting that it may be appropriate to record oximetry for multiple, rather than for a single night (Lewis CA et al., 2003). NOD may be a predictor of PH and cor pulmonale in COPD patients with daytime normal blood oxygen levels (Fletcher EC et al., 1989). However there is no sufficient information about the potential difference between the patients with NOD in development of PH and cardiopulmonary complications. Electrocardiography, chest x-ray, right heart catheterization, MRI, myocardial perfusion scintigraphy, radionuclide angiography, and TTE can be used to asses right heart failure (Wiedemann HP et al., 1997).

There may be a slow increase in PAP in the course of the disease in stable COPD patients. Exercise induced PH appears clinically in early stages but subsequently it will occur even at rest in further stages of COPD. PH usually occurs as a comorbidity in patients with COPD and daytime hypoxemia (PaO_2 <55 or 60 mmHg). It is suggested that long term oxygen therapy is useful to prevent NOD and related complications (Douglas NJ et al., 1998). NOD is typically used as desaturation greater than 3% for at least 5 minutes during sleep period. However, the criteria for the oxygen therapy is contraversial in patients with NOD and without day time hypoxemia because previous investigations used different basal SaO_2% level and nocturnal time spent with SaO_2<90% to define it. Little et al., (1999) defined NOD as a decrease in SaO_2 ≥4% according to basal oxygen saturation level during at least ≥5 minutes. Baldwin et al.,(1995) defined NOD as a decrease in % SaO_2 >5% only and Fletcher et al. (1989) used the criteria that decrease in SaO_2 < 90% together with ≥5 minutes during sleep period. Many of the studies recommended to give nocturnal oxygen instead of continous support to COPD patients with TST_{90} >30% of all sleep time as previously mentioned by Levi-Valensi et al., (1990) and Gorecka et al (1997).

We measured PAP levels by doppler echocardiography and PH diagnosis was detected in 25 (45%) of 55 patients. When we compare PH and non-PH groups, the mean nocturnal oxygen saturation levels and the ratio of nocturnal desaturation time to total sleep time was found to be different and statistically significant (p<0,0001). In PH group; the mean nocturnal SaO_2 level was 91,60±2,17 (%) and desaturation ratio was 22,07±18,60 (%). These measures were 94,25±1,48 (%) and 4,74±7,37 (%) in nonPH group respectively. PH and the thickness of the right ventricle showed a correlation (p<0.0001). Right ventricular hypertrophy is one of the main results of chronic PH and it was shown by some studies that there was a significant correlation between autopsy results and right ventricular thickness showed by echocardiography in COPD patients (Prakash R and Matsukubo H, 1983). However some of the studies did not reveal a correlation, due to increased retrosternal air space especially in emphysematous COPD patients (Murphy ML, 1987). If echocardiography shows a right ventricle wall greater than 5 mm, it is considered as right ventricular hypertrophy. In our study, 10 patients had right ventricular hypertrophy and 9 of these patients (90%) had PH.

DLCO results were also found to be different according to PH existence. Pulmonary vasoconstriction and destruction of the pulmonary parenchyma, especially in the patients with emphysema, results in increase in pulmonary resistance (Mac Nee W, 1994). For this reason, a decrease in DLCO, a functional parameter of pulmonary parenchyma destruction, was an expected finding in COPD and PH. In our study, the DLCO results of the groups with and without PH were 58,28±22,28 and 75,75±18,34 respectively (p=0.009). PH was detected in 6 of 26 (23%) patients with DLCO test ≥75% and in 7 of all 7 (100%) patients with DLCO test ≤40%. According to these results, in the patients with DLCO ≤40%, the probablity of PH is very high. COPD patients with these features sould be investigated with doppler echocardiography or cardiac catheterisation.

As a result of this study PH was detected in 11 of 12 (92%) patients with ≥20% NOD and in only 1(5%) of 20 patients with ≤4% NOD at total sleep time . The mean PAP values were statistically different between the patients with ≥10% NOD (27,81±8,20 mmHg) and <10% NOD (17,82±8,35 mmHg) (p<0,0001). These results may suggest that PH development in COPD patients without day time hypoxemia directly proportional to the NOD period. Eventhough, nocturnal oxygen supporting therapy is indicated in the patients with COPD and NOD ≥30%, we suggest to decrease that level to 20%. Because we detected PH in 92% of these patients.

Echocardiograhic findings and monitoring of nocturnal oxygen saturation levels showed that pulmonary steam velocity, as a marker of pulmonary hypertension, increases in patients having more frequent and longer NOD episodes among the patients with COPD. The most important consequence of nocturnal hypoventilation could be a reduced life expectancy. So an early NOD observation on COPD

patients may be a diagnostic tool to predict PH and to influence survivals. To improve the prognosis of the patients with COPD, an earlier long term oxygen supporting therapy and providing nocturnal blood oxygen levels in normal ranges is essential.

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