Prevalence and profile of sleep-disordered breathing and obstructive sleep apnea in patients with interstitial lung disease at the pulmonary medicine department of a tertiary care hospital in Mumbai

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ABSTRACT

Background: Sleep-disordered breathing (SDB), predominantly obstructive sleep apnea (OSA), is a frequent phenomenon in interstitial lung disease (ILD) and may be associated with significant morbidity and mortality. Methodology: A prospective, observational, hospital-based study was conducted in a tertiary care hospital after ethics committee permission. The study group consisted of 100 consecutive ILD patients diagnosed by a multidisciplinary diagnosis. They were evaluated for the prevalence of SDB with a polysomnography after a comprehensive history, detailed clinical examination, calculation of various pretest probability scores, and relevant prerequisite workup. Results: Out of the total 100 ILD patients, 44 were male (44%) and 56 were female (56%). SDB was present in 57 (57%) patients. Of these, 29 (29%) were found to have only nocturnal oxygen desaturation (NOD), while 28 (28%) had OSA. The 28 cases of OSA were distributed as 15 mild OSA (53.57%), 10 moderate OSA (35.71%), and 3 severe OSA (10.71%). The patients were divided into the following four groups: total study Group (A), patients with OSA (Group B), patients with NOD without OSA (Group C), and no SDB (Group D). The mean forced vital capacity values predicted in the four groups were 53.67%, 50%, 45.56%, and 57.87%, respectively. The mean body mass index in the four groups was 24.56, 27, 26.98, and 24.89 kg/m², respectively. The mean 6-min walk distance in the four groups was 280.7, 250, 256.65, and 311.4 m, respectively. The mean partial pressure of oxygen in the four groups was 65.65, 60, 62.10, and 75.66 mmHg, respectively. The mean apnea-hypopnea index in the study group was 2.98/h, 8.6/h with mild OSA, 21.69/h with moderate OSA, 48.78/h with severe OSA, 3.89/h in patients having NOD without OSA, and 2.54/h in patients with no SDB. Conclusion: SDB in ILD is associated with a significant impact on the cardinal determinants of functional capacity, lung function, and quality of life.

KEY WORDS: Interstitial lung disease, obstructive sleep apnea, sleep-disordered breathing

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INTRODUCTION

Interstitial lung disease (ILD) constitutes a heterogeneous group of disorders that affect the alveolar capillary membrane and lung interstitium, thereby causing a diffusion impairment.^[1] This leads to a chronic hypoxia and restrictive lung disease with ventilatory impairment. They eventually culminate in pulmonary hypertension (PH), cor pulmonale, and respiratory failure. Sleep-related breathing disorders (SRBDs) represent a group of physiopathological conditions that are characterized by an abnormal respiratory pattern during sleep that can be isolated or can coexist with other respiratory, nervous, cardiovascular, or endocrine diseases. SRBDs are now known to be widely prevalent in the general population and contribute to numerous problems resulting from the underlying fragmented sleep patterns. Obstructive sleep apnea (OSA) is a subtype of SRBDs, which is characterized by a repetitive pattern of upper airway collapsibility, airflow obstruction, and resultant arousals. It is associated with repeated episodes of partial or full cessation of breathing during sleep, usually accompanied by oxyhemoglobin desaturation.^[2] Patients with ILD are likely to be at risk for sleep-disordered breathing (SDB) due to limitations in their gas exchange and the ventilatory impairment.^[3] This SDB in ILD can encompass a broad spectrum ranging from nocturnal oxygen desaturation (NOD) alone or NOD associated with OSA of varying intensity. The treatment options available are limited and focus more on halting disease progression rather than definitive cure.^[4] SDB, particularly OSA, constitutes a major comorbidity associated with ILD. The suboptimal sleep quality in ILD is a complex interplay of medical, psychological, social, and therapy-related factors. The basic pathogenesis involves a fragmented sleep architecture, altered respiratory mechanics, nocturnal desaturation, and nocturnal hypoventilation.^[5] This poor sleep quality in ILD has manifold repercussion on the health-related quality of life.^[6] However, the literature available on the prevalence and pattern of SDB in ILD is limited, with majority of studies focusing on idiopathic pulmonary fibrosis (IPF) only.^[7] Hence, there is a need for more research in this deficiently fathomed area.^[8] We, therefore, conducted this study to decipher the prevalence and profile of SDB and OSA in patients with ILD at the pulmonary medicine department of a tertiary care hospital in Mumbai, Maharashtra, India.

METHODOLOGY

A prospective, observational, hospital-based study was conducted in the pulmonary medicine department of a tertiary care hospital after ethics committee permission over a period of 2 years from October 2015 to October 2017. The study group consisted of 100 consecutive patients of age groups >18 years, who attended our outpatient department for clinical symptoms suggestive of ILD and who were diagnosed as cases of ILD based on a multidisciplinary diagnosis. A detailed history including history of presenting symptoms and history to identify known causes of ILD was obtained. A detailed sleep history and a history pertaining to the symptoms of OSA were taken, namely, snoring and its intensity, presence of choking or witnessed breathing pauses, recurrent awakenings from sleep, excessive daytime sleepiness (measured by using the Epworth Sleepiness Scale score), nonrefreshing sleep, increased irritability, and lapses in concentration. It also included prior medical history, especially systemic hypertension, diabetes mellitus, ischemic heart diseases, history of comorbidities such as gastroesophageal reflux diseases, thyroid-related disorders, history of addictions such as smoking or alcohol consumption, and medication history. The physical examination included measurement of height; weight; body mass index (BMI) (kg/m²); neck, waist, and hip circumferences; and waist/hip ratio. Investigations done before sleep study included blood investigations, namely, complete blood count; fasting sugar levels; arterial blood gas (ABG) analyses; a two-dimensional echocardiography; 6-min walk test; and spirometry to measure forced vital capacity (FVC), forced expiratory volume in first second (FEV1), and FEV1/FVC ratio. Ear, nose, and throat examination with Mallampati pharyngeal grading system was done. Before sleep study, pretest probability of OSA was assessed in all patients using clinical prediction rules such as ESS score, SACS score, APNEIC score, STOP-Bang Scoring Model, and Berlin questionnaire. The patients were then subjected to a polysomnography (PSG) with a limited channel sleep study which was either hospital- or home-based depending on the preference of the patient as per the clinical guidelines for the use of unattended portable monitors in the diagnosis of OSA in adult patients given by the American Academy of sleep Medicine (AASM) 2007.^[9] This limited sleep study machine recorded continuous polygraphic recordings for nasal and oral airflow (thermistors), tracheal sounds, thoracic and abdominal effort, limb movements, body position, and oxyhemoglobin saturation (pulse oximeter). A night time ABG was also done at the time of sleep study. The definitions of apnea, hypopnea, OSA, and the severity of OSA were as per the diagnostic criteria given by the 2007 AASM guidelines.^[10] For SDB, the criteria of the ICSD-2 for the diagnosis of sleep-related breathing disorders in idiopathic pulmonary fibrosis (IPF) and diffuse parenchymal lung disease (DPLD) were used. These criteria are adopted from those concerning the SDB disorders in chronic obstructive pulmonary disease.^[10] Statistical analysis was done with Statistical Package for the Social Sciences 15 (SPSS-15). Data were analyzed in the form of percentage and frequencies. P < 0.05 was considered statistically significant.

RESULTS

Our study group consisted of 100 consecutive patients of ILD, of which 44 were male (44%) and 56 were female (56%). Minimum age of ILD case in our study group was 28 years and maximum age was 80 years, with a mean age of 56.44 years. The most common age group affected was 51-60 years with a female predominance. Out of 100 patients with ILD, the most common type of ILD in our study group was hypersensitivity pneumonitis present in 36 cases (36%) followed by IPF in 19 cases (19%), connective tissue disease-related ILD in 15 cases (15%). sarcoidosis in 11 cases (11%), nonspecific interstitial pneumonia in 11 cases (11%), combined pulmonary fibrosis and emphysema in 4 cases (4%), silicosis in 2 cases (2%), and respiratory bronchiolitis ILD in 2 cases (2%), as shown in Table 1. Out of 100 cases, 57 cases (57%) were diagnosed to have NOD. Out of these, 29 cases (29%) were found to have only NOD, while 28 cases (28%) had OSA. Forty-three cases (43%) were found to have normal sleep study, as shown in Table 2 and Figure 1. The number of patients having NOD with or without OSA in different forms of ILD has been shown in Table 3 and Figure 2. Out of the 28 cases of OSA diagnosed among the 100 ILD patients, 15 were having mild OSA (53.57%), 10 were having moderate OSA (35.71%), and 3 had severe OSA (10.71%), as shown in Table 4. The patients were divided into the following four groups: total study group (A), patients with OSA (B), patients with NOD without OSA (C), and no SDB (D), and parameters namely mean FVC, mean BMI, mean 6-min walk distance (6MWD), mean partial pressure of oxygen (PaO₂), and mean apnea-hypopnea index (AHI) were studied.

It was found that the mean FVC in the study group was 53.67% predicted, 50% predicted in patients with OSA, 45.56% predicted in patients having NOD without OSA, and 57.87% predicted in patients without SDB. The difference was statistically significant with P = 0.48 (<0.5). The mean BMI in the study group was 24.56 kg/m², 27 kg/m² in patients with OSA, 26.98 kg/m² in patients having NOD without OSA, and 24.89 kg/m² in patients without SDB. The difference was statistically significant with P = 0.26. The mean 6MWD in the study group was 280.7 m, 250 m in patients with OSA, 256.65 m in patients having NOD without OSA, and 311.4 m in patients without SDB. The difference was however not statistically significant with P = 0.67. The mean PaO₂ in the study group was 65.65 mmHg, 60 mmHg in patients

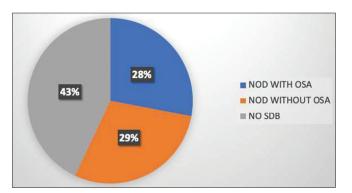


Figure 1: Distribution of sleep-disordered breathing in the study group

Table 1:	Types of in	terstitial	lung diseas	e in	the study	
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Types	Male	Female	Total (%)	Cumulative percent
HP	13	23	36 (36)	36
IPF	10	9	19 (19)	55
NSIP	5	6	11 (11)	66
CTD-related	3	12	15 (15)	81
ILD				
Sarcoidosis	5	6	11 (11)	92
CPFE	4	0	4 (4)	96
Silicosis	2	0	2 (2)	98
RBILD	2	0	2 (2)	100
Total	44	56	100 (100)	

ILD: Interstitial lung disease, HP: Hypersensitivity pneumonitis, IPF: Idiopathic pulmonary fibrosis, NSIP: Nonspecific interstitial pneumonia, CTD: Connective tissue disease, CPFE: Combined pulmonary fibrosis and emphysema, RBILD: Respiratory bronchiolitis interstitial lung disease

 Table 2: Distribution of sleep-disordered breathing in

 the study group

SDB	Frequency (%)	Cumulative percent		
NOD without OSA	29 (29)	29		
OSA	28 (28)	57		
No SDB	43 (43)	100		
Total	100 (100)			

NOD: Nocturnal oxygen desaturation, OSA: Obstructive sleep apnea, SDB: Sleep-disordered breathing

Table 3: Distribution of sleep-disordered breathing in	I
each type of interstitial lung disease in the study grou	up

Diagnosis		Total			
	NOD with OSA	NOD without OSA	NO SDB	DB	
HP	9	13	14	36	
IPF	7	7	5	19	
NSIP	5	2	4	11	
CTD-related ILD	5	2	8	15	
Sarcoidosis	1	1	9	11	
CPFE	2	1	1	4	
Silicosis	0	1	1	2	
RBILD	0	1	1	2	
Total	29	28	43	100	

ILD: Interstitial lung disease, HP: Hypersensitivity pneumonitis, IPF: Idiopathic pulmonary fibrosis, NSIP: Nonspecific interstitial pneumonia, CTD: Connective tissue disease, CPFE: Combined pulmonary fibrosis and emphysema, RBILD: Respiratory bronchiolitis interstitial lung disease, NOD: Nocturnal oxygen desaturation, OSA: Obstructive sleep apnea, SDB: Sleep-disordered breathing

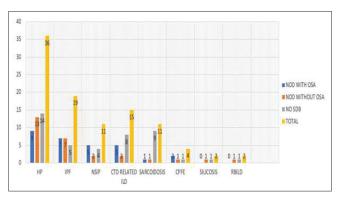


Figure 2: Distribution of sleep-disordered breathing in each type of interstitial lung disease in the study group

with OSA, 62.10 mmHg in patients having NOD without OSA, and 75.66 mmHg in patients without SDB. The difference was statistically significant with P = 0.39. The mean AHI in the study group was 2.98/h, 8.6/h with mild OSA, 21.69/h with moderate OSA, 48.78/h with severe OSA, 3.89/h in patients having NOD without OSA, and 2.54/h in patients without SDB. The mean pulmonary artery systolic pressure (PASP) in the study group was 30.5 mmHg, 38.5 mmHg in patients with moderate and severe OSA, respectively, 35.5 mmHg in patients having NOD without OSA, and 25.0 mmHg in patients without SDB. The difference was statistically significant with P = 0.39 [Table 5 and Figure 3].

DISCUSSION

This study was undertaken with an endeavor of determining the prevalence, profile, and characteristics of SDB and OSA in patients with ILD. SDB and OSA is a commonplace but comparatively underreported phenomenon in ILD. The predisposition for upper airway

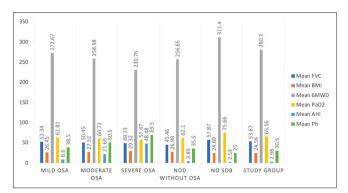


Figure 3: Comparison between mean forced vital capacity, mean body mass index, mean 6-min walk distance, mean partial pressure of oxygen, mean apnea-hypopnea index, and mean pulmonary hypertension in the study group

Table 4: Grading of obstructive sleep apnea

Grades (AHI/h)	Cases
No (0-5)	72
Mild (6-15)	15
Moderate (16-30)	10
Severe (>30)	3

AHI: Apnea-hypopnea index

collapsibility due to the underlying restrictive lung disease and ventilator control imbalance due to intermittent hypoxia have been postulated as the causative factors for this phenomenon^[11]. SDB in ILD has been reported earlier in various studies, with emphasis majorly laid on the IPF subgroup. SDB in ILD can be largely classified into standalone NOD or NOD with OSA component. In our study group, out of 100 cases, 57 cases (57%) were diagnosed to have SDB. Twenty-nine cases (29%) was found to have only NOD, while 28 cases (28%) had NOD with OSA, while the rest 43 cases (43%) were found to have normal sleep study. The prevalence of OSA in ILD has been variably reported in literature in earlier studies. Aydoğdu et al. reported OSA in 24 out of 65 patients with ILD. They found that hypopneas, and not apneas, were the predominant respiratory abnormality in these patients.^[12] In a prospective study by Mermigkis *et al.*^[13] conducted a study on newly diagnosed IPF patients in which OSA was reported in 20 of 59 patients. Pihtili et al.^[14] presented polysomnographic data in an assorted group of ILD patients in a prospective study. OSA was present in 34 (Includes IPF, sarcoidosis and scleroderma) 68(All ild patients having osa other than above 3) IPF 14 (82) means 14 patients had osa out of total 82 patients of IPF enrolled. Turner *et al.*^[15] studied 83 consecutive patients with sarcoidosis seen over a 6-week time period. A total of 14 sarcoid patients (17%) were found to have sleep apnea, which was significantly higher than the control group with 3/91 (3%, P < 0.001). However, in our study, out of 11 patients of sarcoidosis, only 1 patient was diagnosed as OSA (9.1%). However, all the above studies have studied the prevalence of only OSA in either ILD or IPF. The prevalence of NOD, however, has been studied in relatively fewer studies. Perez-Padilla et al.^[16] reported lower mean nocturnal saturation (SpO₂) among ILD patients as compared to age- and gender-matched controls. In their a correlation between lowest awake SpO₂ and NOD, Bye et al.^[17] reported that patients with ILD develop marked NOD particularly during REM sleep. The NOD in ILD has been hypothesized as a spillover effect of the transient or sustained daytime hypoxia with a substantial cumulative effect.^[11] This can result from various contributing factors including ventilation perfusion mismatch, alveolar hypoventilation, and dependent small airway closure, with OSA being a contributory factor in some cases. Hence, it is pertinent to understand that NOD can occur

Table 5: Comparison between mean forced vital capacity, mean body mass index, mean 6-min walk distance, meanpartial pressure of oxygen, and mean apnea-hypopnea index in the study group

	Study group (100)	NOD With OSA (2		OSA (28)		NOD Without OSA (29)	No SDB (43)
		Mild	Moderate	Severe	Mean		
Mean FVC%	53.67	52.34	50.45	49.23	50	45.56	57.87
Mean BMI kg/m ²	24.56	26.45	27.52	29.32	27	26.98	24.89
Mean 6MWD (m)	280.7	272.67	258.98	230.76	250	256.65	311.4
Mean PaO ₂ (mmHg)	65.56	62.81	60.73	57.47	60	62.10	75.66
Mean AHI (h)	2.98	8.6	21.69	48.78	17.32	3.89	2.54
Mean PH (mmHg)	30.5	38.5	50.5	69.5	43.8	35.5	25.0

FVC: Forced vital capacity, BMI: Body mass index, 6MWD: 6-min walk distance, AHI: Apnea-hypopnea index, PH: Pulmonary hypertension, Pa0₂: Partial pressure of oxygen, NOD: Nocturnal oxygen desaturation, OSA: Obstructive sleep apnea, SDB: Sleep-disordered breathing

in association with or independent of OSA. Deciphering this phenomenon is also crucial for deciding about the chief modality of therapy, i.e., continuous positive airway pressure (CPAP) versus nocturnal oxygen therapy in ILD patients.^[18] Patients with ILD having standalone NOD as a type of their SDB benefit more from nocturnal oxygen therapy, whereas patients with OSA as their chief SDB benefit more from CPAP therapy.

SDB, either OSA or NOD, has the potential to cast an impact on the cardinal determinants of the functional capacity in ILD patients. The cardinal determinants can be studied in the form of mean 6MWD, PaO₂, and FVC. We attempted to analyze the correlation between the trends of these variables in the subsets of our study group. The patients were divided into the following four groups: total study group, patients with OSA, patients with NOD without OSA, and no SDB, and these parameters were studied. The mean FVC in the study group was 53.67% predicted, 50% predicted in patients with OSA, 45.56% predicted in patient having NOD without OSA, and 57.87% predicted in patients with no SDB. The difference was statistically significant with P = 0.48 (<0.5). Thus, the mean FVC was significantly lower in the SDB group in both OSA and NOD without OSA subsets. The mean 6MWD in the study group was 280.7 m, 250 m in patients with OSA, 256.65 m in patients having nocturnal desaturation without OSA, and 311.4 m in patients without SDB. Though the 6MWD was also lower in the OSA and NOD subsets, this difference was not statistically significant with P = 0.67. The mean PaO_a in the study group was 65.65 mmHg, 60 mmHg in patients with OSA, 62.10 mmHg in patients with NOD without OSA, and 75.66 mmHg in patients with no SDB. The difference was statistically significant with P = 0.39. The mean BMI in the study group was 24.56 kg/m², 27 kg/m² patients with OSA, 26.98 kg/m² in patients having NOD without OSA, and 24.89 kg/ m^2 in patients with no SDB. The difference was statistically significant with P = 0.26. Thus, it is evident that SDB in the form of both OSA and NOD has a pessimistic impact on the cardinal determinants of the functional capacity As evident by the statistically significant lower FVC, lower PaO₂, higher bmi and though not statistically significant but lower 6mwd were observed in these groups as compared to patients with no SBD.

Even after an extensive literature search, we did not come across work done pertaining to the correlation of these parameters with SDB in ILD patients. However, there have been certain studies done which gauge the impact on these variables in IPF patients having OSA. Mermigkis *et al.*^[19] in their study on 18 IPF patients assessed with nocturnal PSG found that AHI was positively correlated with BMI and negatively correlated with FEV1 and FVC percentages. They hypothesized that an increased BMI and a significant impairment in pulmonary function testing may be predictors of OSA in this population. This observation was similar to our study. However, in a study done by Lancaster *et al.* on fifty patients with IPF, it was found that spirometry, lung volumes, and DLCO did not inversely correlate with the AHI or the severity of SDB. In addition, BMIs in patients did not strongly correlate with their AHI.^[20] In another study done on 41 patients with IPF, poor sleep quality and increased daytime sleepiness were documented. However, these parameters were not significantly associated with age, gender, body mass index, or lung function factors.^[21] The mean PASP in the study group was 30.5 mmHg, 38.5 mmHg in patients with mild OSA, 50.5 and 69.5 mmHg in patients with moderate and severe OSA, respectively, 35.5 mmHg in patients having NOD without OSA, and 25.0 mmHg in patients without SDB. The difference was statistically significant with P = 0.39. We thus found that PASP was significantly higher in ILD patients with SDB, especially in those with OSA. Research pertaining to PH in ILD patients confirms the fact that there is a link between the severity of NOD and the gravity of PH both on echocardiography and right heart catherization.^[22] The underlying mechanism is based on an increased expression of a vasoactive peptide endothelin-1 in patients with ILD during the periods of NOD.^[23] This can indirectly be inferred from the evidences that nocturnal oxygen supplementation in ILD not only improves the quality of life and symptomatology but also has an optimistic impact on the pulmonary hemodynamics.^[18]

In a progressive disease like ILD where the therapy options are limited and the benefits of therapy are not substantial, pulmonary rehabilitation and optimum management of comorbidities forms the cornerstone of management.^[24] The overall repercussions of SDB, both NOD and OSA on ILD, are manifold in terms of affection of symptomatology, disease progression, quality of life, and survival. Hence, its timely recognition and management is essential to improve the health-related quality of life and also to reduce the mortality. Our study is the first of its kind done on a large sample size in ILD patients consisting of a heterogeneous cohort of various ILDs, focusing on the prevalence of SDB and its subtyping, and also going further to gauge their impact on the cardinal determinants of the functional capacity. This highlights the significance of the association, which paves a way for further research in the area.

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Conflicts of interest

There are no conflicts of interest.

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