RESEARCH	ARTICLE

Association of Smoking Status with COPD in North Indian Population

Sarika Pandey[1], Rajiv Garg[1*], Surya Kant[1], Priyanka Gaur[2], Ajay Verma[3], Prashant Mani Tripathi[1], Rajeev Kumar[1]

[1]Department of Respiratory Medicine, King George's Medical University, Lucknow, Uttar Pradesh, India
[2]Department of Physiology, King George's Medical University, Lucknow, Uttar Pradesh, India
[3]Department of Pulmonary & Critical Care Medicine, King George's Medical University, Lucknow, Uttar Pradesh, India

*__Address for Correspondence:__ Dr. Rajiv Garg, Professor, Department of Respiratory Medicine, King George's Medical University, Lucknow- 226010, Uttar Pradesh, India

ABSTRACT- Background- The chronic obstructive pulmonary disease is a chronic inflammatory disease and a leading cause of morbidity and mortality worldwide. Smoking is a major risk factor in COPD. Smoking damages the air sacs, airway and the lining of the lungs and due to this lung have trouble moving enough air in and out making hard to breathe. Smoking may act as a trigger factor for many people who have COPD and can either cause an exacerbation or flare-up of symptoms. The present study aims to determine the association of smoking status with different stages of COPD, and clinical symptoms in a North Indian population.

Methods- The present study was conducted on 160 stable COPD patients in the department of Respiratory Medicine, King George Medical University, Lucknow. The study subjects included were diagnosed cases of stable COPD of both genders.

Results- Out of 160 patients enrolled there were 41.8% smokers, 24.3% non-smokers, and 33.7% ex-smokers. The present study found a significant association ($p<0.02$) of smoking status with different stages of COPD, although non-significant association ($p=0.96$) was observed between smoking status and clinical symptoms.

Conclusion- The significant association of smoking status was observed with different stages of COPD, while the non-significant association was observed with clinical symptoms in the present study in north Indian population. Smoking cessation will be helpful in reducing the progression and management of this disease in smokers.

Key-words- Clinical symptoms, Gold stage, Chronic Obstructive pulmonary disease, Smoking

INTRODUCTION

Chronic Obstructive Pulmonary Disease is a chronic inflammatory disease and a leading cause of morbidity and mortality worldwide [1]. It is a common, preventable and treatable disease, characterized by persistent respiratory symptoms and airflow limitation that is due to airway and/or alveolar abnormalities usually caused by significant exposure to noxious particles or gases. As per World Health Organization (WHO), three million people die from COPD each year [2]. It is estimated that more than 90% of COPD deaths occur in low and middle income countries [3]. COPD continues to be an important public health problem in India. It is independently associated with low-grade systemic inflammation, with a different inflammatory pattern than that observed in healthy subjects [4].

Systemic inflammation in COPD patients has been associated with increased neutrophil, macrophage, T-lymphocytes and high concentrations of inflammatory mediators in peripheral blood such as C-reactive protein (CRP) and other cytokines (IL-6, IL-8 and TNF-α etc) [5-10]. Breathlessness, cough and/or sputum production are the most important respiratory symptoms associated with this disease [11].

Cigarette smoking is the most studied risk factor in COPD [12]. Smoking may act as a trigger factor for many people who have COPD and can either cause an exacerbation or flare-up of symptoms. Smoking damages the air sacs, airway and the lining of the lungs and due to this lung have trouble moving enough air in and out making hard to breathe. Long term smoking causes airway inflammation characterized by neutrophil, macrophage and activated T lymphocyte infiltration and by increased CRP and cytokine concentration. Occupational exposures, environmental exposure and indoor air pollution from biomass cooking are the other risk factors for the development and the progression of COPD. Exacerbations and Co-morbidities also contribute to the overall severity in individual patients. The present study was done in north Indian population to determine the association of smoking status with different stages of COPD and clinical symptoms.

Access this article online

Quick Response Code	Website: www.ijlssr.com
	crossref DOI: 10.21276/ijlssr.2018.4.2.12

MATERIALS AND METHODS

Study population and selection of subjects- The present study was carried out in the department of respiratory medicine, King George medical university, Lucknow a tertiary care hospital, North India. The study subjects included were diagnosed cases of stable COPD of both genders. COPD patients (n=160) were enrolled from the OPD of the respiratory department. The subjects were residing in the geographic area of northern India. The study was approved by the institutional ethical committee and written informed consent was obtained from all the subjects. The diagnosis of COPD was based on pulmonary function test, which was done in all patients. According to GOLD criteria, COPD was defined on the basis of the post bronchodilator FEV1/FVC ratio of less than 0.70 and reversibility to an inhaled bronchodilator in FEV1 <12% or <200ml after administration of 200 μg Salbutamol (2 puffs) using a pressurized metered dose inhaler with a spacer. All patients, who were included free from any disease or exacerbation forth preceding 2 months. Subjects reporting with a history of pulmonary tuberculosis, cardiac diseases, ILD, pregnancy, diabetes, and cancer were excluded from the study. Patients with any other systemic disease other than COPD were also excluded. In all the subjects, body weight and height were noted and body mass index (BMI) was calculated according to the formula of weight in kilograms divided by the height in meter square. A detailed clinical history of respiratory symptoms was also obtained.

Based on the patient self-reported smoking history, subjects were classified into three groups:

Group 1: Current smokers

Group 2: Ex-smokers (Ex-smokers were those who didn't smoke from last one year. Pack-years were calculated as (number of cigarettes smoked per day × number of years smoked)/20)

Group 3: Never-smokers

Statistical Analysis- Graph pad PRISM version 6.01 was used for analysis of data. Values have been represented in mean±SD (in case of continuous variable) and expressed as number and percentages (in case of categorical variables). Chi-square test was used for comparison of categorical data. P-value <0.05 was considered statistically significant.

RESULTS

Demographic history- The baseline characteristics of the patients are shown in Table 1. Age of patients ranged from 35 to 75 years. Mean age of patients was 56.96±9.64. Among all study subjects, 129 (80.6%) were males and 31 (19.4%) females. The mean BMI of patients was 20.32±4.10.

Table 1: Demographic profile of stable COPD patients

Parameters	COPD patients (N=160)
Age (yrs)	56.96±9.64
Height (cm)	159.3±10.56
Weight (kg)	51.44±10.68
BMI (kg/m^2)	20.32±4.10
Gender	
Male	129(80.62%)
Female	31(19.37%)
Spirometry parameters	
Post FVC (L)	2.05±0.64
Post FEV1 (L)	1.13±0.41
Post FEV1/FVC	54.26±10.13
Post FEV1% pred	44.04±14.91

Smoking History- The present study comprises 67 (41.8%) smokers (Group 1), 39(24.3%) non-smokers (Group 2) and 54 (33.7%) ex-smokers (33.7%). Among non-smokers, 23 patients were having a history of exposure to biomass. The mean pack-years was (20.29±17.35) in the COPD patients. The number of people, who smoke bidi (72.7%) was greater in our study in comparison to cigarette (20.5%) while there were fewer people, who smoke both (6%).
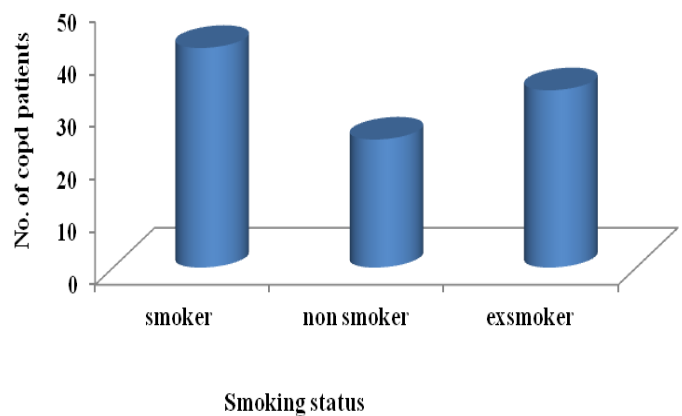

Fig. 1: Distribution of COPD patients according to smoking status

Spirometric values such as mean FEV 1% predicted FVC and FEV1/FVC ratio of COPD patients are mentioned in Table 1. According to GOLD criteria, COPD patients were grouped into four stages based on their severity. There were 2 patients (1.25%) having mild COPD (stage 1), 48 patients (36.25%) have moderate COPD (stage 2), 57 patients (35.6%) have severe COPD (stage 3), while 53 patients (33.1%) were having very severe COPD (Fig. 2).
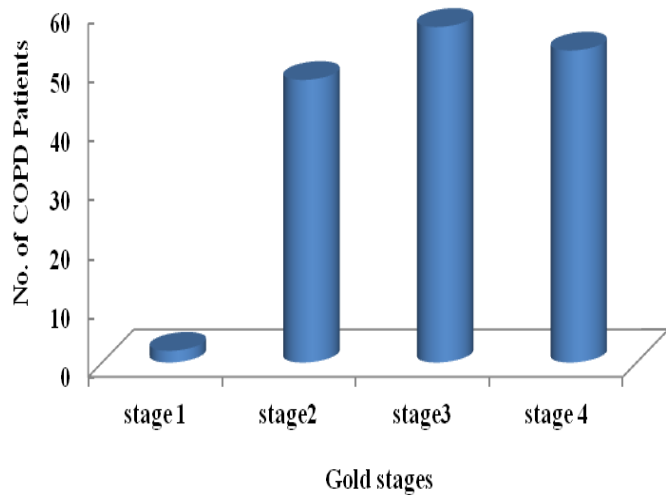
Fig. 2: Distribution of COPD patients into different GOLD stages on the basis of severity

Fig. 4: Distribution of COPD patients on the basis of smoking status according to presenting clinical symptoms

COPD patients had been divided into three groups (Fig. 3) smokers, non-smokers and ex-smokers on the basis of severity-

Group 1: Among smokers, there were 1 mild COPD patient, 18 moderate COPD, 17 severe COPD and 31 very severe COPD.

Group 2: Among non-smokers there was no patient with mild COPD, 15 moderate COPD, 19 severe COPD and 5 very severe COPD patients.

Group 3: Among ex- smokers there were 1 mild COPD patient, 15 moderate COPD, 21 severe COPD and 17 very severe COPD patients. Statistically significant higher no of smokers were observed in very severe patients (stage 4) of COPD (p value<0.02).
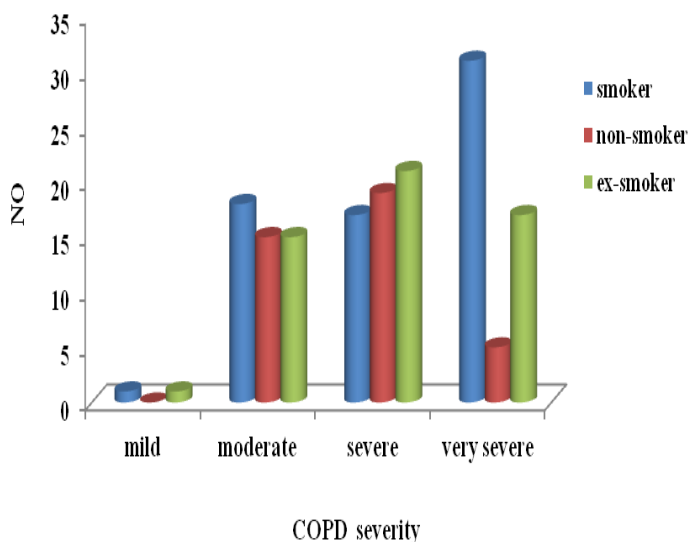
On the basis of smoking status, clinical symptoms observed in 3 different groups are shown in Fig. 4. In the present study, there were 67 smokers, 39 non-smokers, and 54 ex-smokers.

In Group 1, Breathlessness was the main symptom observed in 66 patients (98.5%) followed by a cough in 63(94%), chest pain in 15(22.4%) and wheezing in 13 patients (19.4%). Total 14 patients (21 %) also complained for fever, 11(16%) for loss of sleep and 22(32%) for loss of appetite.

In Group 2, Breathlessness was the main symptom observed in 39 patients (100%) followed by cough in 38 (97.4%), wheezing in 12 (30.7%) and chest pain in 6 patients (15.3%). Total 6 patients (15.3%) also complained about fever, 7(17.5%) for loss of sleep and 7 (17.5%) for loss of appetite.

In Group 3, Breathlessness was the main symptom observed in 54 patients (100%) followed by cough in 52 (96.3%), chest pain in 11(20.3%) and wheezing in 9 patients (16.6%). Total 9 patients also complained about fever, 8 for loss of sleep and 10 for loss of appetite.

We do not find any significant association between the clinical symptoms and smoking status (p=0.96) but significant association was observed between stages of COPD and smoking status in our study (p value 0.02).

Fig. 3: Distribution of smokers, non-smokers and ex-smokers on the basis of severity

Clinical Symptoms- Breathlessness (95.6%) and cough (93.1%) were the most prominent symptoms observed in more than 90% patients followed by wheezing (22.5%) and chest pain (18.75%). Fever (17.5%) loss of sleep (16.2%). loss of appetite (24.3%) was the other symptoms.
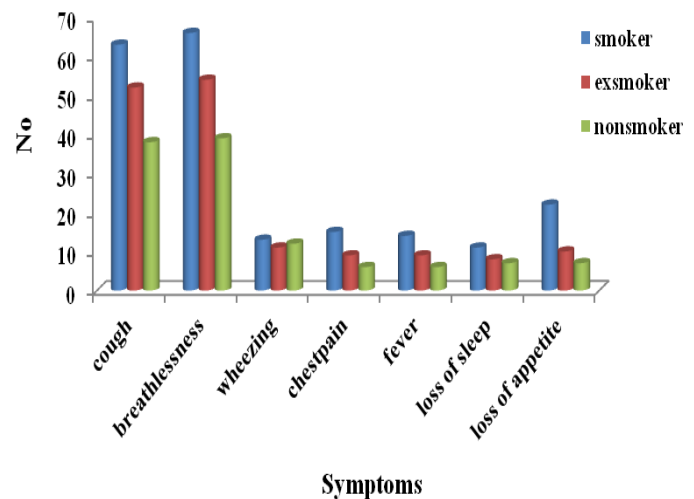
DISCUSSION

Tobacco smoking had been recognized as the most important risk factor for chronic obstructive pulmonary [13]. Tobacco smoke contains in excess of 4000 chemicals in each puff and more than 70 cancer-causing chemicals or carcinogens [14]. In previous studies, it has been seen that subjects, who were current and past smokers were at an increased risk of having COPD in comparison with those who were never smokers [15,16].

Data from recent study also shows that the proportion of COPD was statistically higher (*P*<0.05) in current smokers and those exposed to more pack-years of tobacco smoke, mixed smokers, those exposed to ETS or having an occupational exposure to dust/fumes/smoke for longer duration, subjects using biomass fuels [17]. In the population-based survey of adults with a smoking history,

prolonged tobacco use was associated with an increased likelihood of having COPD and it was also seen that former smokers who had quit smoking for nearly 10 years had a lower prevalence of COPD and respiratory symptoms than current smokers [18]. The results of the present study also show higher no of smokers COPD patients 41.8% smokers and 33.7% ex-smokers. Highest no of smokers were observed in stage 4 of COPD while higher no of ex-smokers was present in stage 3 of COPD in this study. We observed a significant association between smoking status and different stages of COPD (p<0.02).

It has been shown in previous studies that biomass exposure also contributes to a significant proportion of COPD, which was inconsistent with our study [19]. We found that among 39 non-smoker patients, 24 gave a history of biomass exposure. Previous studies showed that among non-smokers, there was a substantial body of epidemiological evidence, which links occupational exposure to dust, gases/vapors, and fumes with chronic airflow obstruction and with nearly 15–20% attributable risk in the substantial population. According to previous studies in India [20], the indoor air gets polluted due to smoke from combustion of solid fuels such as dried dung, wood and crop residue used for cooking and heating, especially in villages and it is responsible for having COPD. We also studied the association of smoking status with clinical symptoms of COPD patients and found a non-significant association (p value=0.96). Smoking cessation has been seen to be the most effective strategy for slowing or halting the progression of the disease [21,22]. Smoking cessation improved respiratory function and prevents the excessive decline in lung function in smokers with COPD as well as in smokers without chronic symptoms [23,24].

CONCLUSIONS

Studies on smokers with COPD shown that lifelong smokers have a 50% probability of developing COPD during their lifetime and there were some evidences that the risk of developing COPD falls by about half with smoking cessation. The current study found a significant association of smoking status with different stages of COPD, although the non-significant association was observed between smoking status and clinical symptoms. Smoking cessation will be helpful in reducing the progression of COPD, if proper counselling of patients is done by the physician.

ACKNOWLEDGMENT

REFERENCES

[1] Lopez A, Shibuya K, Rao C, Mathers C, Hansell A, Held L, et al. Chronic obstructive pulmonary disease: current burden and future projections. ERSJ, 2006; 27: 397–412.

[2] World Health Organization, Burden of COPD, 2005.

[3] World Health Organization. Chronic obstructive pulmonary disease (COPD) Fact sheet No 315, World Health Organization, 2011.

[4] Garcia-Rio F, Miravitlles M, Soriano JB, Munoz L, Duran-Tauleria E, Sanchez, et al. EPI-SCAN Steering Committee: Systemic inflammation in chronic obstructive pulmonary disease: a population-based study. Respiratory Research, 2010; 11: 63.

[5] Gan W, Man S, Senthilselvan A, Sin D. Association between chronic obstructive pulmonary disease and systemic inflammation: a systematic review and meta-analysis. Thorax, 2004; 59: 574-80

[6] Agusti AG, Noguera A, Sauleda J, Sala E, Pons JXB: Systemic effects of chronic obstructive pulmonary disease. Eur. Respir. J., 2003; 21: 347-36.

[7] Franciosi LG, Page CP, Celli BR, Cazzola M, Walker MJ, et al. Markers of disease severity in chronic obstructive pulmonary disease. Pulm. Pharmacol. Ther., 2006; 19: 189-99.

[8] Takabatake N, Nakamura H, Abe S, Hino T, Saito H, et al. Circulating leptin in patients with chronic obstructive pulmonary disease. Am. J. Respir Crit Care Med., 1999; 159: 1215-19.

[9] Pinto-Plata VM, Mullerova H, Toso JF, Feudjo-Tepie M, Soriano JB, et al. C- reactive protein in patients with COPD, control smokers and non-smokers. Thorax, 2006; 61: 23-28.

[10] Higashimoto Y, Yamagata Y, Taya S, Iwata T, Okada M, et al. Systemic inflammation in chronic obstructive pulmonary disease and asthma: Similarities and differences. Respirol., 2008; 13: 128-33.

[11] Pandey S, Garg R, Kant S, Gaur P, Singh S, Singh P. C - reactive protein as a Biomarker in Chronic Obstructive Pulmonary Disease Patients: A Mini Review. Int. J. Life Sci. Scienti. Res., 2018; 4(1): 1534-35.

[12] Mannino DM et al. COPD: epidemiology, prevalence, morbidity and mortality, and disease heterogeneity .Chest, 2002; 121: 121S-26S.

[13] Donald P, Tashkin A, Robert P. Murray B. Smoking cessation in chronic obstructive pulmonary disease Respiratory Med., 2009; 103: 963-74.

[14] Pryor WA. Cigarette smoke radicals and the role of free radicals in chemical carcinogenicity. Environ Health Perspect., 1997; 105(S4): 875–82.

[15] Ke-Sheng Wang, Liang Wang, Shimin Zhen, Long-Yang Wu. Associations of Smoking Status and Serious Psychological Distress with Chronic Obstructive Pulmonary Disease. Int. J. High Risk Behaviors and Addiction, 2013; 2(2): 59-65.

[16] Shahab L, Jarvis MJ, Britton J, West R Prevalence, diagnosis and relation to tobacco dependence of chronic obstructive pulmonary disease in a nationally representative population sample. Thorax, 2006; 61: 1043-47.

[17] Sinha B, Vibha, Singla R, Chowdhury R. An epidemiological profile of chronic obstructive pulmonary disease: A community-based study in Delhi. J. Postgrad. Med., 2017; 63: 29-35.

[18] Liu et al. Smoking duration, respiratory symptoms, and COPD in adults aged 45 years with a smoking history Int. J. COPD, 2015; 10: 1409–16.

[19] Meldrum M, Rawbone, R, Curran, AD Fishwick D. The role of occupation in the development of chronic obstructive pulmonary disease (COPD). Occup. Environ. Med., 2005; 212-14.

[20] Jindal SK, Gupta D, Aggarwal AN. Guidelines for management of chronic obstructive pulmonary disease (COPD) in India: a guide for physicians (2003). Indian J. Chest Dis. Allied Sci., 2004; 46: 137-53.

[21] Anthonisen NR, Connett JE, Kiley JP, et al. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV1. The lung health study JAMA, 1994; 272: 1497-1505.

[22] Scanlon PD, Connett JE, Waller LA, Altose MD, Bailey WC, Buist AS. Smoking cessation and lung function in mild-to-moderate chronic obstructive pulmonary disease. The lung healthstudy Am. J. Respir. Crit. Care Med., 2000; 161: 381-90.

[23] Anthonisen NR, Connett JE, Murray RP. Smoking and lung function of Lung Health Study participants after 11 years. Am. J. Respir Crit. Care Med., 2002; 166(5): 675–79.

[24] Willemse BW, Postma DS, Timens W, Ten Hacken NH. The impact of smoking cessation on respiratory symptoms, lung function, airway hyperresponsiveness and inflammation. Eur. Respir. J., 2004; 23(3): 464–76.