



ORIGINAL RESEARCH PAPER

Pulmonology

Study of Epidemiology and Effect of Smoking in Patients with Obstructive Lung Diseases

KEY WORDS: Asthma, COPD, smoking

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ABSTRACT

Obstructive airway diseases, both chronic obstructive pulmonary disease (COPD) and asthma are leading cause of mortality and morbidity worldwide. Active or passive smoking is being recognized as most important causative factor for COPD. There is no direct relationship of smoking as a cause of asthma in most of the adult patients, however there is enough of evidence for adverse effects of smoking on asthma too. 1,2 There are a few reports on smoking relationship with different forms of atopy and asthma. As there is paucity of data, we planned this study to assess the epidemiology of smoking in patients with obstructive lung diseases in India.

Our study population had a male predominance with maximum number of patients in age group of 40-49 years. 60% patients were active smokers, while 29.3% were passive smoker. 48.9% patients were former smokers. 54% patients were bidi smoker while 6% were cigarette smokers. COPD patients had a higher smoking exposure than patients with asthma. Exposure to Environmental Tobacco Smoke (ETS), was also a significant factor for development of COPD and asthma especially in females.

Introduction

COPD, asthma and other chronic respiratory diseases are the second leading cause of death in the population aged 25–69 years as per Indian census (2001-2003). 3 Compared to USA and Europe, COPD accounts for four times more death in India.4

COPD is listed as the significant cause of morbidity and mortality worldwide,5 however, this chronic disease is barely even acknowledged in the health statistics of many countries. Tobacco smoking, either active or passive is the most common cause of chronic airway diseases such as chronic bronchitis (CB) and COPD.6 One of the important risk factor for developing COPD is air pollution which can result from the burning various type of fuels.7

Smoking cessation is single most effective intervention to reduce the risk of developing COPD and also to prevent its progression as lifelong smokers have a 50% probability of developing COPD during their lifetime.7 The exact role of smoking cessation on the airway inflammation process in COPD is still unknown.

Asthma is a chronic inflammatory disorder of the airways with variable airflow obstruction and airway hyper-responsiveness which leads to recurrent episodes of wheezing, breathlessness, chest tightness and coughing. It is often reversible either spontaneously or with treatment.7

Despite of no direct relationship of smoking as a cause of asthma, it can be influenced in one or other way by either active smoking or by exposure to environmental tobacco smoke (ETS) i.e. passive smoking. Smoking also plays an important factor in the development of airway remodelling, fixed airway obstruction and an exaggerated lung function decline. Smoking can adversely affect the health and treatment-outcomes of asthma. Smoking cessation as well as control of ETS-exposure should therefore constitute important and essential components of asthma therapy also.8,9,10,11, This study was planned to assess the epidemiology of smoking in patients with obstructive lung diseases.

Material and Methods
Study Design (Fig-1)

Study was carried out as a single center prospective and cohort study conducted at LRS Institute of Tuberculosis and Respiratory Diseases, New Delhi; which is a tertiary care, referral and teaching institute, during the period from 1st June 2008 to 15th March 2010 (Total of 150 patients were selected).

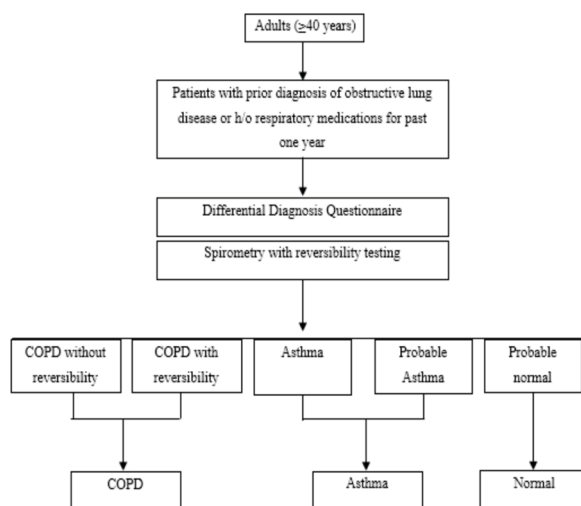


Figure 1. Algorithm depicting study design

Subject Recruitment

Patients were recruited with age ≥40 years along with prior evidence of obstructive lung disease (e.g. COPD, asthma) on physician diagnosis or with h/o respiratory medications within the past year, regardless of smoking status. Informed consent was taken. Patients with history of known preexisting or concomitant non-obstructive lung disease, active tuberculosis, unstable heart disease and with acute respiratory illness were excluded.

Study Tool

Questionnaire included questions covering sociodemographic data and smoking history. Special emphasis was given to the correct documentation of the smoking status which was recorded as smoking index. All interviews were performed by the same investigator in order to eliminate inter observer variability. Questions were translated in Hindi language according to standard protocols for better understanding and ease of answering. Spirometry with reversibility testing was done. Patients were classified as per Table-1. Observations were compared to each group to obtain relevant results.

Table-1: Criteria establishing the Study Diagnosis

Study diagnosis	Criteria
COPD without reversible component	Post bronchodilator FEV1/FVC <0.70 Reversibility <200 ml or <12% of baseline FEV1
COPD with reversible component	Post bronchodilator FEV1/FVC <0.70 Reversibility ≥200 ml and ≥12% of baseline FEV1
Asthma	Post bronchodilator FEV1/FVC ≥0.70 Reversibility ≥200 ml and ≥12% of baseline FEV1
Probable asthma	Post bronchodilator FEV1/FVC ≥0.70 Reversibility <200 ml or <12% of baseline FEV1 and Prior diagnosis of asthma or subject is taking corticosteroids on a chronic basis
Probable normal	Post bronchodilator FEV1/FVC ≥0.70 Reversibility <200 ml or <12% of baseline FEV1 and Does not fulfill criteria for probable asthma

Data was analysed using univariate analysis for non-numerical values and ANOVA for numerical values. p<0.05 was considered statistically significant.

Results and observation

150 patients were enrolled for the study. Our study population had a male predominance, there being 84 males (56%) and 66 females (44%). Out of 150, maximum number of patients, 66 (44%) were found in age group 40-49 years consisting of 32 males and 34 females. Minimum numbers of patients, 11 (7.3%) were found in the age group 70-79 years consisting of 9 males and 2 females. The mean age of male patients was 51.75±4.64 years while of female patients was 50.73±9.61 (Table-2).

Table-2: Relationship between patient age and sex

Age Group	Male (n=84)	Female (n=66)	Total (n=150)
40-49	32 (38.1%)	34 (51.6%)	66 (44%)
50-59	29 (34.5%)	16 (24.2%)	45 (30%)
60-69	14 (16.7%)	14 (21.2%)	28 (18.7%)
70-79	9 (10.7%)	2 (3%)	11 (7.3%)
Mean	51.75±4.64	50.73±9.61	50.06±9.81

On the basis of spirometry, study diagnosis of COPD without reversible component was made in 49 (32.7%) patients. Study diagnosis of COPD with reversible component was made in 45 (30%) patients. Study diagnosis of asthma was made in 18 (12%) patients. Study diagnosis of probable asthma and probable normal was made in 18 (12%) patients and in 20 (13.3%) patients respectively. Out of 150, maximum number of patients, 94 (62.7%) had diagnosis of COPD, 36 patients (24%) had diagnosis of asthma while diagnosis of probable normal was made in 20 (13.3%) patients.

Out of 150 patients, 90 patients (60%) were active smokers (current and former), 44 patients (29.3%) were passive smoker and 16 patients (10.7%) were non-smoker.

Regarding active smoking, out of 90, 24 (16%) patients were current smoker, 66 (44%) patients were former smoker. It was observed that there were smaller differences in the proportion of current smokers (16% in COPD versus 19.4% in asthma). COPD patients had highest proportion of former smokers (48.9%) as compared with other groups (Table-3).

Table-3: Relationship between smoking status and diagnosis

		COPD (n=94)	Asthma (n=36)	Probable normal (n=20)	Total (n=150)	P value
Smoking status	Current	15 (16%)	7 (19.4%)	2 (10%)	24 (16%)	0.438 (NS)

	Former	46 (48.9%)	12 (33.3%)	8 (40%)	66 (44%)
	Never	33 (35.1%)	17 (47.2%)	10 (50%)	60 (40%)

Out of 90 active smokers, 81 (54%) patients were bidi smoker while 9 (6%) patients were cigarette smokers. Bidi smoking was more common (61.6% and 41.7 in COPD and asthma respectively) as compared to cigarette smoking (4.3% and 11.1 in COPD and asthma respectively) and thus type of smoking was not found to be statistically significant (p value = 0.19) in differential diagnosis of COPD and asthma in our study.

Smoking index was 0-500 in 131 (87.3%) patients, 501-1000 in 17 (11.3%) patients and 1001-1500 in 1 (0.7%) patient with a mean of 195.33±288.02. COPD patients had a higher smoking exposure (mean smoking index of 259.8) than patients with asthma (mean smoking index of 87.8). Thus in our study, smoking index differentiates COPD from asthma with higher smoking exposure correlating with diagnosis of COPD (p=0.02) (Table-4).

Table-4: Relationship between type of smoking, smoking index and diagnosis

		COPD (n=94)	Asthma (n=36)	Probable normal (n=20)	Total (n=150)	P value
Smoking Index	0-500	77 (82%)	34 (94.4%)	20 (100%)	131 (87.3%)	0.002 (S)
	501-1000	15 (16%)	2 (5.6%)	0 (0%)	17 (11.3%)	
	1001-1500	1 (1%)	0 (0%)	0 (0%)	1 (0.7%)	
	1501-2000	1 (1%)	0 (0%)	0 (0%)	1 (0.7%)	
	Mean	259.79 ±331.98	87.78 ±151.12	86 ±118.15	195.33 ±288.02	
Type of Smoking	Bidi	57 (60.6%)	15 (41.7%)	9 (45%)	81 (54%)	0.19 (NS)
	Cigarette	4 (4.3%)	4 (11.1%)	1 (5%)	9 (6%)	
	None	33 (35.1%)	17 (47.2%)	10 (50%)	60 (40%)	

Regarding passive smoking, out of 150, 31 (20.7%) were exposed to bidi/cigarette or both, 25 (16.7%) were exposed to chullah, 18 (12%) were exposed to bidi/cigarette and chullah and 76 (50.6%) were not exposed to passive smoking.

65.1% females in the study group were exposed to biomass fuels. Exposure to biomass fuel was found to be more common in asthmatics (33.3%) and normal (35%) groups as compared to COPD (25.5%) due to more number of females in both these groups. However, exposure to bidi/cigarette passive smoke was more common in COPD patients than asthma (23.5% Vs 13.4%). Thus in our study, biomass fuels have been identified as one of the major risk factors for development of COPD and asthma especially in females. However, history of passive smoking was not statistically significant (p value = 0.8) in differentiating COPD from asthma in our study (Table-5).

Table-5: Relationship between various types of passive smoking and diagnosis

		COPD (n=94)	Asthma (n=36)	Probable normal (n=20)	Total (n=150)	P value
Passive Smoking	Bidi/Cigarette or both	22 (23.4%)	5 (13.9%)	4 (20%)	31 (20.7%)	0.8 (NS)

Chulla	13 (13.8%)	7 (19.4%)	5 (25%)	25 (16.7%)
Bidi/ Cigarette and Chulla	11 (11.7%)	5 (13.9%)	2 (10%)	18 (12%)
None	48 (51.1%)	19 (52.8%)	9 (45%)	76 (50.6%)

Discussion

According to the World Health Organization (WHO), one hundred million deaths were caused by tobacco abuse in the 20th century. WHO also stressed that if current trends will continue, the death toll will go up to one billion in the 21st century and it will keep rising by more than eight million deaths per year. Developing countries are at higher risk.

Either active or passive smoking is being recognized as most important causative factor for development of obstructive lung disease.6 Air pollution due to burning of various types of fuels are also other important risk factor.7

Bidi/cigarette smoke contains an extremely high concentration of oxidants which induce inflammation in the lung and its airway.12 Most of the smokers develop some respiratory impairment during their life.13,14 Lundback B etal studied the prevalence and risk factors for COPD. They found that the 50% of smokers eventually develop COPD as per GOLD definition. This finding is of major clinical significance as prevalence of COPD is only 8% and 14% as per BTS and GOLD guidelines respectively.15

COPD is still unknown as inflammatory and structural changes in the airways increase with disease severity and persist despite smoking cessation. Van Schayck, etal, in his editorial mentioned that despite the ongoing inflammatory process, the rate of development of COPD can be reduced when patients at risk of developing the disease stop smoking.16

Epidemiology of COPD is not well studied in India and there are many unanswered questions because of paucity of systematically collected prevalence data. Spirometry is the internationally accepted gold standard for the diagnosis of COPD. Even available prevalence estimates are not based on spirometry testing or they have adopted non-standard methods/protocols.17

Continued smoking by adult asthmatics is the likely cause of irreversibility of airway obstruction. Such patients behave like those of COPD. This is in consistence with the Dutch hypothesis, first postulated in 1961 that COPD developed due to exaggerated airways damage from smoking exposure of patients with underlying atopic diagnosis.

There is paucity of data on to support the smoking association of asthma in adults. Recent studies from India done on large populations have strongly supported the smoking association of asthma in adults.17,18

We did this study to assess the epidemiology of smoking in patients with obstructive lung diseases.

Our study population had a male predominance. We failed to find any epidemiological studies on relation of sex with COPD and asthma in India.

In our study, maximum number of patients were in age group of 40-49 years. Minimum numbers of patients, 11 (7.3%) were found in the age group 70-79 years. In an Indian study on epidemiology of asthma and chronic bronchitis (CB), Jindal SK etal found the prevalence of asthma and CB as 2.05% (adults aged ≥15 years) and 3.49% (adults aged ≥35 years) respectively. They also found that advancing age, smoking, household environmental tobacco smoke exposure, asthma in a first-degree relative, and use of unclean cooking fuels were associated with increased odds of asthma and CB.17 In contrast to our study,

Shahab etal, in his study from England, found that there was an approximately linear increase in the prevalence of COPD with age, irrespective of smoking status.19

We did spirometry to make a study diagnosis of COPD, asthma and probable normal in patients with pre-existing physician diagnosis of obstructive lung disease. Although spirometry is the internationally accepted gold standard for the diagnosis of COPD, however we failed to found available prevalence estimates based on spirometry.

In our study 60% patients were active smokers (16% current and 44% former), while 29.3% were passive smoker. Proportion of current smokers were almost same in COPD and asthma. Former smokers (48.9%) were higher in COPD as compared with other groups. In a similar study it was found that the prevalence of COPD among current smokers (22.4%) and former smokers (24.6%) were higher than the prevalence among non-smokers (7.0%).20 In another study, it was found that current cigarette smoking was significantly higher among people with COPD (34.9%, 95% CI 32.1 to 37.8) than among those without COPD (22.4%, 95% CI 21.4 to 23.4). While the majority of people with COPD were former or never smokers, the prevalence of current cigarette smoking in people with spirometry-defined COPD was higher than in the general population.19

Another population based study (Tasmanian Longitudinal Health Study) on a cohort born in 1961 reports the development of fixed airway obstruction in middle age contributed by active smoking and current clinical asthma.21

In our study, out of 90 active smokers, 54% patients were bidi smoker while 6% were cigarette smokers. Bidi smoking was more common (61.6% and 41.7 in COPD and asthma respectively) as compared to cigarette smoking (4.3% and 11.1 in COPD and asthma respectively). The Indian study on Epidemiology of Asthma, Respiratory symptoms and Chronic bronchitis (INSEARCH) demonstrated odds ratios of 1.82 for cigarette and 2.87 for bidi smoking in a sample population of 169575 adults of over 15 years of age.17

There was also a clear dose relationship of number of asthma symptoms with number of cigarettes smoked.22 There was also a dose response relationship of number of cigarettes with impairment of lung function assessed by spirometry performed at baseline and at 18 months follow-up.23

In our study, COPD patients had a higher smoking exposure (mean smoking index of 259.8) than patients with asthma (mean smoking index of 87.8). In his study, H Park etal also demonstrated that individuals with ≥20 pack-years exhibited a 3-fold increased prevalence of COPD as compared to non-smoking individuals after adjusting for sex and age.20 In another study, Perret JL etal, concluded that the association of fixed airway obstruction was equivalent to a 33 pack-year history of smoking for late onset current clinical asthma compared to 24 pack-year history for late onset clinical asthma.21

In our study, exposure to Environmental Tobacco Smoke (ETS), was also a significant factor for development of COPD and asthma especially in females. Passive exposure to cigarette smoke may also contribute to the development of COPD by increasing the lung total burden of inhaled particles and gases.24 Passive smoking affects asthma in a similar fashion. ETS exposure in adult asthmatics from smoking by spouses, siblings or colleagues is troublesome. Asthmatic symptoms sharply decline after the ETS exposure is reduced.2

The role of indoor air pollution caused by exposure to biomass fuel in poorly ventilated dwellings is perfectly established as a risk factor for the development of COPD, being one the most important determinants of the disease, especially among women in developing countries.25,26

Smoking cessation is single most effective intervention to reduce the risk of developing COPD but the exact role of smoking cessation on the airway inflammation process is unknown.

These results have implications for the recognition, prevention, and treatment of COPD in primary and secondary care. Increasing awareness of COPD in the general population and specifically among smokers would aid the early diagnosis of this disease.

In conclusion, smoking exposure is known to influence the development of obstructive lung disease as an important risk-factor. Smoking cessation as well as adoption of measures to avoid ETS-exposure are essential steps for the comprehensive and effective management of these diseases.

Limitations

The size of the study population is small. We need large scale multicentric studies with a bigger sample size. The study included participants ≥ 40 years of age which may underestimate the diagnosis of asthma as asthma is more prevalent in younger age group.

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