

Outcome And Risk Factors Of Chronic Obstructive Pulmonary Disease (COPD) In Smokers Vs. Non-Smokers

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Abstract

Background:

The chronic obstructive pulmonary disease (COPD) is a progressive lung illness characterized by an obstructed breathing and a regular inflammation. Although smoking is the leading risk factor, some significant COPD patients are nonsmokers, and thus other factors would be likely to contribute towards the ailment.

Objectives:

The purpose of this study has been to estimate differences in clinical outcomes, as well as to identify specific COPD risk factors for smokers as compared to even non-smokers.

Study Design: A Retrospective Comparative Study.

Place and Duration of Study. Department of Pulmonology Jinnah Teaching Hospital Peshawar. from jan 2023 to jan 2024

Methods:

A retrospective cohort study was conducted using data from 200 COPD patients, divided as smokers and non-smokers. Demographics, clinical presentation, radiology and spirometry were the data studied. Exacerbation incidence, hospitalisations, reported patient QoL, and annual mortality were studied in the 1-year follow-up.

Results:

Smokers had less FEV1 ($42.1 \pm 13.5\%$) and were more prone to exacerbations (mean 3.2/year) compared to non-smokers (FEV1 $54.7 \pm 11.8\%$; exacerbations 1.9/year; p The rate of exposure of non-smokers to indoor air pollution, biomass, and secondhand smoke was higher. The mortality rate for smokers was 15%, far greater than that for non-smokers (7%).

Conclusion:

Smoking is still an important cause of COPD however, non-smokers have similar vulnerabilities to COPD from environmental and workplace exposures. Smokers generally have worse clinical outcome although smokers alone and non-smokers need specific preventive measures.

Keywords

COPD, Smoking, Non-smoker, Pulmonary outcomes, Air pollution, Risk factors

Introduction

Chronic Obstructive Pulmonary Disease (COPD) is a common, slowly-developing respiratory disease characterized by continued respiratory obstruction that usually cannot be fully reversed. It accounts for the third leading cause of death globally according to the World Health Organization (WHO) and one of the main causes of the morbidity and mortality worldwide [1]. Patients with COPD undergo significant negative impact on the quality of life, major costs of healthcare services and increased risk of premature death. While cigarette smoking is the main modifiable risk factor for COPD, many studies suggest that significant numbers of patients will present with COPD even without active smoking [2-3]. Studies show that in the case of developing countries, 25-45 percent of the COPD cases are diagnosed in nonsmokers [4]. Such a trend is particularly prominent in the case of women and those who reside in the countryside, where constant contact with indoor air pollutants, such as burning biomass fuel, occupational dust, and passive smoking occurs [5]. In addition, genetic predisposition, and prior respiratory infections like tuberculosis, and environmental conditions have also been associated with non-smokers' development of COPD [6]. The presentation of COPD, its severity and progression will vary for smokers and non-smokers. Attraction of smoking has a more pronounced airflow impairment, more acute flare-ups, faster loss of lung function consequent to its cytotoxic effect on the respiratory epithelium [7]. In non-smokers, the clinical picture of COPD is usually milder, though this is not obligatory and some individual patients can have significant morbidity due to particular exposure to non-tobacco pollutants [8]. Despite these differences, relatively few studies have been conducted to compare risk factors and outcome for COPD in smoking and non-smoking subjects. Appreciated of such variations will be essential to achieving precision healthcare and creating more precise public health strategies. Considering the high prevalence of both tobacco use and indoor air pollution in low- and middle-income countries like Pakistan, such investigations are suitably relevant [9]. The main goal of our study was to examine and compare the clinical outcomes and factors of risk for COPD among smokers and non-smokers. Our expectation was that smokers would show worse disease severity and less desirable outcomes compared to non-smokers. However, non-smokers were expected to have a distinctive pattern of exposure to environmental and occupational hazards. It is by doing this comparison that we hope to give useful insights for clinicians and policy makers as it relates to the overall causes of copd and to illustrate the significance of put in place comprehensive prevention strategies.

Methods

It was a retrospective comparative study conducted in the Department of Pulmonology Jinnah Teaching Hospital Peshawar from Jan 2023 to Jan 2024. Medical records of 200 COPD patients were analyzed by grouping them into smokers, i.e. n=130 and non-smokers, i.e. n=70. With COPD diagnosis being according to the Global Initiative for Chronic Obstructive Lung Disease (GOLD) criteria with a post-bronchodilator FEV₁/FVC ratio less than <0.70>. The study obtained patients' demographic information, pack-years of smoking, spirometry data, and clinical information about exacerbations, hospitalizations, and mortality relating to environmental exposure to biomass fuel, passive smoking, and occupational dust. Quality of life at baseline in the study was assessed through the COPD Assessment Test (CAT). The study was given ethical clearance by the institutional review board. All data pertaining to patients were anonymized to maintain their privacy and privacy of patient information.

Inclusion Criteria

Eligible patients were aged 40 years and above, had an established COPD diagnosis conforming to GOLD criteria, and they had a comprehensive clinical and spirometric record for the period of the study.

Exclusion Criteria

Outpatient exclusion, if a patient had asthma-COPD overlap, active TB, lung cancer, major heart conditions, or medical records missing, was needed to reduce potential confounding.

Data Collection

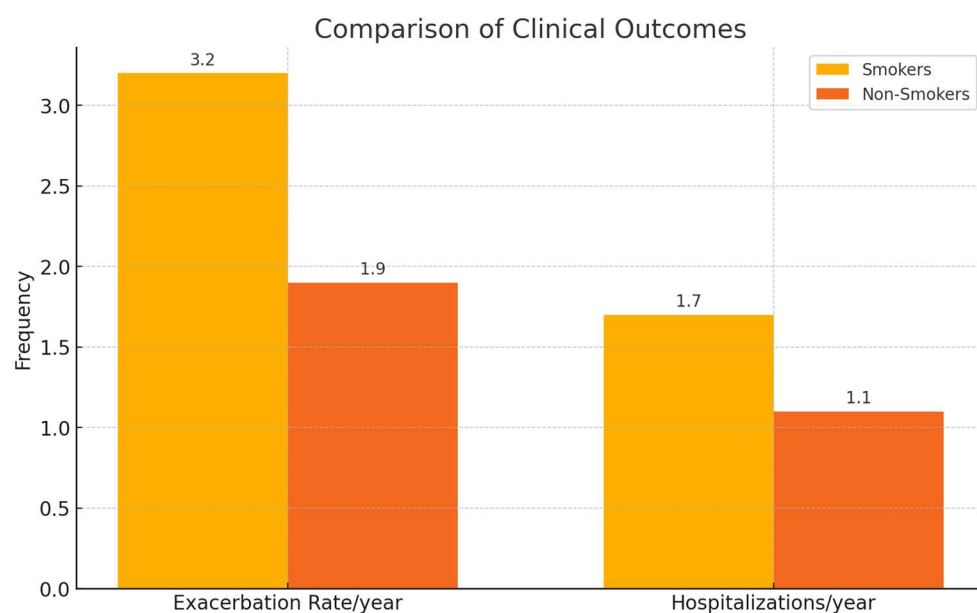
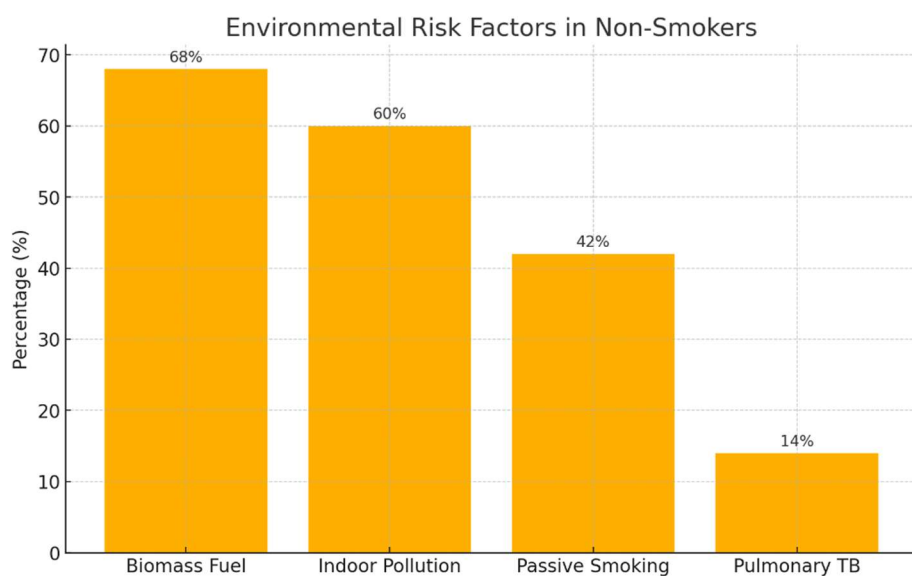
Patient information was extracted from the electronic medical records system of the hospital using structured data extraction forms. Age, gender, smoking history, spirometry data, exposure to environmental factors, frequency of exacerbations, hospitalization rates, CAT scores, and one-year mortality outcomes were included in the data.

Statistical Analysis

Statistical analyses were conducted using SPSS version 24.0. Demographic variables were summarized using descriptive statistics. Continuous characteristics were measured using independent t-tests, and categorical variables were measured using chi-square tests. Statistical significance in all comparisons between smokers and non-smokers was considered when p value was under 0.05.

Results

Of the 200 COPD patients investigated, 65% of the 130 patients identified themselves as smokers and 35% of the 70 patients were non-smokers. The average age of the smokers was 66.5 ± 9.2 years while the non-smokers had a mean age of 63.8 ± 8.5 years ($p=0.08$). A majority of the identified smokers were males 78.5% while the non-smokers had females 60% as the major group. Smokers had a significantly lower mean FEV₁ than the non-smokers ($42.1 \pm 13.5\%$ vs $54.7 \pm 11.8\%$) ($p<0.001$). The mean annual number of exacerbations was much higher in the smokers (3.2 events/year) than in non-smokers (1.9 events/year, $p=0.01$). Similarly, smokers had increased rates of hospitalisation (mean 1.7/hly) compared to non-smokers (mean 1.1/hly). Non-smokers had a high level of risk factors, namely indoor air pollution (60%), biomass fuel (68%), passive smoking (42%), and treated pulmonary tuberculosis (14%). Smokers had very high CAT scores compared to non-smokers (mean 24.3 ± 5.7 compared to mean 18.5 ± 4.9 in non-smokers) which was statistically significant ($p<0.05$). The mortality rate within a year among smokers was 15% compared to 7% for nonsmokers. These outcomes highlight a more severe disease course in smokers and demonstrate a significant disease burden and unique risk profiles of non-smokers.

**Table 1: Baseline Demographic Characteristics**

Variable	Smokers (n=130)	Non-Smokers (n=70)	p-value
Number of Patients	130	70	-
Mean Age (years)	66.5	63.8	0.08
Male (%)	78.5%	40.0%	<0.01

Female (%)	21.5%	60.0%	<0.01
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Table 2: Clinical and Spirometric Outcomes

Variable	Smokers	Non-Smokers	p-value
FEV ₁ (%)	42.1	54.7	<0.001
Exacerbation Rate/year	3.2	1.9	0.01
Hospitalizations/year	1.7	1.1	0.03
CAT Score	24.3	18.5	0.02
Mortality (%)	15%	7%	0.04

Table 3: Environmental Risk Factors in Non-Smokers

Risk Factor	Prevalence in Non-Smokers (%)
Biomass Fuel Exposure	68%
Indoor Air Pollution	60%
Passive Smoking	42%
Previous Pulmonary TB	14%

Discussion

Our research highlights significant differences both in clinical outcomes and risk factors for COPD among smokers and non-smokers. The findings of our study support the fact that smokers with COPD have greater airflow reduction, more frequent exacerbations, worse quality of life, and increased risk of death compared to non-smokers COPD patients. However, the negative effects of diseases in nonsmokers, particularly due to indoor air contaminates and second hand smoke, remains significant and clinically meaningful. Following our findings, a multicenter study by Salvi and Barnes revealed that in developing countries, COPD in non-smokers has exceeded 40%, mainly due to biomass smoke exposure, which initiates chronic airway inflammation and reformation similar to that of the tobacco smoke [9]. Hu et al. also found out that exposure to indoor air pollution from cooking with solid fuels is also a major independent risk factor for COPD in nonsmoking women, more common in rural areas [10]. As evidenced by the observation of a large majority of non-smoking patients being female, with many reporting regular use of biomass fuels and exposure to unventilated kitchens, our findings lunch other. In accordance with our results, Lamprecht et al. using data from the BOLD research reported that COPD non-smokers undergo relatively little spirometric abnormalities, but have strong symptom burdens, and poor QOL scores [11]. Our findings showed that non-smokers had greater mean FEV₁ values and fewer hospitalizations, but their sensitivity to other environmental risks was critical to the onset of the disease. Smokers had higher outbreak rates and mortality rates than the non-smokers, an observation that follows the reports as shown by Hogg et. al. who in turn reported increased small airways damage and emergence of emphysema in

smoking induced-copd patients [12]. Furthermore, studies show that smokers have a reduced effectiveness in response to bronchodilators and corticosteroids in a form of corticosteroid resistance and continuous oxidation stress that accelerates the disease development [13]. Studies by Eisner et al. showed that adult passive smoking increases risk of COPD by 60% in non-smokers, and it is another piece of evidence of the crucial role of secondhand smoke in non-smoker COPD cases [14]. In addition, many chronic pulmonary illnesses such as tuberculosis are known to play a substantial role in stunting airflow in non-smokers [15]. This 14% TB history found in non-smoking COPD patients is consistent with research by Byrne et al. reporting that approximately 20-30% of high-burden countries' COPD cases develop in post-TB [16]. Exposure history has recently been tipped as an important factor by Global Initiative for Chronic Obstructive Lung Disease (GOLD), emphasizing the need to diversify the concept of COPD risk [17]. Through the use of improved environmental settings, alternative fuel for cooking and educating the population about COPD it is possible to significantly reduce rates of COPD among non-smokers [18].

Conclusion

smoking causes COPD, which correlates with poorer clinical outcomes; however, non-smokers find themselves in a similar situation with disease burden due to environmental factors. Knowledge pertaining to these particular risk indicators is essential for the creation of effective preventive measures and group-specific management strategies that can reduce disease burden, hospital admissions and death rates in the two groups.

Limitations

Retrospective data obtained from one center was used for the study, thus it may not generalize findings generally. Due to lack of comprehensive environmental exposures documentation and information on such as socioeconomic and genetic influences, results may have been biased. Long-term monitoring only covered one year.

Future Directions

The need for prospective multicenter studies with more extended follow-ups to describe non-smoker COPD phenotypes is required. Investigation of genetic, occupational, and epigenetic possibilities could reveal new risk mechanisms for COPD. It is suggested that future studies should comprise interventions to decrease indoor air pollution and increase awareness among the population at increased risk.

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Authors Contribution

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