A COMPARISON OF CLINICAL PROFILE AND PREVALENCE OF COMORBIDITIES AMONG BIOMASS AND TOBACCO SMOKE-INDUCED COPD PATIENTS AT A TERTIARY CARE CENTRE IN NORTH INDIA

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BACKGROUND

Although, smoking is the most common risk factor implicated in the causation of chronic obstructive pulmonary disease (COPD), biomass smoke exposure is an important risk factor, especially in women residing in rural areas of developing countries such as India. Comorbidities are prevalent in patients with COPD because of systemic inflammation. The prevalence of comorbidities among biomass smoke-induced COPD has not been widely studied.

Aims and Objectives- To assess whether patients exposed to biomass fuel have similar clinical profile and prevalence of comorbidities as smoker COPD or not.

MATERIALS AND METHODS

The clinical characteristics and prevalence of comorbidities were compared between 411 male patients with smoker COPD and 288 female patients with biomass smoke-induced COPD.

RESULTS

The patients exposed to biomass fuel were all females, were younger, had higher body mass index (BMI) and had less severe disease (higher values of post bronchodilator (BD) forced expiratory volume in the first second (FEV1, %)). The mean age in smoker COPD was significantly higher than in biomass smoke-induced COPD (p < 0.001). The mean BMI was lower in smoker COPD as compared to that in biomass smoke-induced COPD and this difference was statistically significant (p= 0.042). Coronary artery disease (CAD) (13.86% versus 3.81%, p < 0.0001) and systemic hypertension (HTN) (15.32% versus 3.47%, p < 0.0001) were significantly more common in smoker COPD without any other significant differences. Obstructive sleep apnoea (OSA), lung cancer and anaemia were found to be more common in biomass fuel-induced COPD, although not statistically significant.

CONCLUSION

CAD and systemic HTN were significantly more prevalent in smoker COPD.

KEYWORDS

Chronic Obstructive Pulmonary Disease, Biomass, Tobacco, Comorbidity.
generates higher concentration of PM\textsuperscript{10} (Respirable particulate matter with diameter ≤ 10 μm) than charcoal.\textsuperscript{3}

Comorbidities are very common in COPD and contribute to overall disease severity. The comorbidities frequently associated with COPD are coronary artery disease (CAD), heart failure (HF), systemic hypertension (HTN), obstructive sleep apnoea (OSA), diabetes mellitus (DM), osteoporosis and anaemia. The presence of comorbidities depends on gender, disease severity and COPD phenotype.\textsuperscript{10} Persistent inflammation is the cause of most of the comorbidities associated with COPD. Also, common risk factors and genetic predisposition play a role in the development of these comorbidities. Since there are pathophysiological differences in tobacco smoke-induced and biomass smoke-induced COPD, we aimed to find whether the prevalence of various comorbidities among these two groups also differ.

**MATERIALS AND METHODS**

This was a retrospective study conducted at the respiratory centre of a tertiary care hospital in Uttarakhand, which caters to a large rural population residing in hilly regions and where biomass fuel is traditionally used for cooking. Patients were evaluated over a period of two years from November 2015 to November 2017. The inclusion criteria were\textsuperscript{[1]}: a diagnosis of COPD established according to GOLD guidelines.\textsuperscript{[2]} Patients were classified as smoker COPD if they had > 10 pack years of tobacco smoking and biomass smoke-induced COPD if they had > 10 years exposure to biomass smoke for cooking purposes. The exclusion criteria were\textsuperscript{[1]}: co-existing respiratory illnesses such as pulmonary or pleural tuberculosis (TB), interstitial lung disease (ILD) or bronchiectasis.\textsuperscript{[3]} Patients with unstable coronary artery disease or arrhythmias. The spirometric parameters recorded for the purpose of the study was post bronchodilator FEV\textsubscript{1}, which denotes disease severity. A questionnaire based protocol was used to record demographic data and symptom profile. Body mass index (BMI) was calculated as weight (in kilograms)/[height]\textsuperscript{2} (in meters). Comorbidities were assessed using previous prescriptions, medication use or a diagnosis established during work at the hospital. The following comorbidities were recorded for all patients: CAD, HF, HTN, DM, lung cancer, OSA and anaemia. Patients with proven CAD on angiography, HF on echocardiography, OSA on polysomnography, histopathological proven lung cancer and DM and anaemia diagnosed through biochemistry were included in the study.

**Statistical Analysis**

Data was analysed using Statistical Package for Social Sciences (SPSS). Values were represented in mean ± standard deviation (SD) and frequencies. For statistical analysis, independent ‘t’ test was used for continuous variables and Chi-square test was used for discrete variables. P is the level of significance, where ‘p’ < 0.05 is statistically significant.

**RESULTS**

A total of 699 patients [411 (58.79%) smokers and 288 (41.20%) biomass smoke exposed] with COPD were included in the study. Table 1 compares the characteristics between the two groups. The patients exposed to biomass fuel were all females, were younger, had higher BMI and had higher values of post bronchodilator (BD) FEV\textsubscript{1}; The mean age in smoker COPD was 62.6 ± 11.0 years, while the mean age in biomass smoke-induced COPD was 59.2 ± 10.98 years and this difference was statistically significant (p < 0.001). The mean BMI for smoker COPD was 22.3 ± 3.99 kg/m\textsuperscript{2}, while that for biomass smoke-induced COPD was 23.08 ± 5.65 kg/m\textsuperscript{2} and this difference was also statistically significant (p = 0.042). There was no significant difference between mean post BD FEV\textsubscript{1} % among the two groups.

Table 2 compares the relative prevalence of comorbidities between the two groups. CAD (13.86% versus 3.81%, p<0.0001) and systemic HTN (15.32% versus 3.47%, p<0.0001) were significantly more common in smoker COPD without any other significant differences. OSA, lung cancer and anaemia were found to be more common in biomass fuel-induced COPD, although not statistically significant.

**DISCUSSION**

It has been documented that an average Indian woman has 60,000 hours of exposure to smoke from biomass fuel in her lifetime.\textsuperscript{11} Behera et al developed a Biomass Exposure Index (BEI) to measure the magnitude of exposure, calculated as the average hours spent cooking per day multiplied by the number of years of cooking.\textsuperscript{12} In a single centre study conducted in South India, Mahesh et al identified a threshold where A BEI ≥ 60 is said to be high risk for developing COPD.\textsuperscript{13} Biomass fuel causes goblet cell metaplasia, increased mucus production and neutrophilic infiltration of bronchial wall.\textsuperscript{14}

We found in our study that biomass exposed COPD patients were younger, had higher BMI and had higher values of spirometric indices. This is similar to the study by Golpe et al who showed that biomass fuel induced COPD has slower decline of FEV\textsubscript{1} and different distribution of phenotypes in
comparison to smoking induced COPD. Similarly, Cheng et al retrospectively analysed 206 patients with tobacco smoke-induced COPD and 81 cases of biomass smoke-induced COPD. They reported that COPD caused by biomass fuel was more common in females, had dyspnoea as more common symptom, had lower BMI and were in Group B or D according to GOLD. No statistically significant difference was found in age, Modified Medical Research Council (MMRC) scale or exacerbation frequency. On the other hand, Camp et al reported that on matching by age and severity of airflow obstruction, biomass fuel exposed females had lower quality of life and more hypoxaemia than females exposed to tobacco smoke.

We also found that CAD and systemic hypertension were significantly less common in biomass induced COPD. No significant association was found among other comorbidities. A similar prevalence was reported by Golpe et al who found that ischaemic heart disease (IHD), peripheral vascular disease and peptic ulcer disease are significantly more common in tobacco smokers than in biomass smokers. No other significant association was found between other comorbidities. In contrast, Cheng et al found that allergic diseases (such as allergic rhinitis and bronchial asthma) were significantly more common in biomass induced COPD in comparison to smoking induced COPD, where lung cancer was more common. There was no significant difference in the cardiac comorbidities such as CAD, HF or arrhythmias. Rivera reported that intimal thickening of pulmonary arterioles causing pulmonary hypertension is more common in patients exposed to biomass smoke than tobacco smoke.

Solleiro-Villaricencio et al have shown that there is predominantly a Th2-type inflammatory response in the body by persistent exposure to biomass fuel smoke. They also found that peripheral blood of biomass exposed individuals have increased interleukin-4 (IL-4) producing T cells in comparison to tobacco smokers who have Th-17, IL-6 and IL-8 in blood. IL-6 is associated with atherosclerosis and increased mortality in COPD, while IL-8 is associated with cerebrovascular disease. This study further highlights the fact that since the pathophysiology of the two groups differ, the prevalence of comorbidities also differ.

This study had certain shortcomings. The possibility of overlap between tobacco smokers and biomass exposed individuals was not taken into consideration. All smoker COPD patients were males, while all non-smoker COPD patients were females, leading to a bias on the basis of gender. Also, due to retrospective nature of the study, cumulative exposure could not be calculated and a dose-response relationship could not be established. Certain significant comorbidities such as osteoporosis, depression and arrhythmias have not been included in our study.

CONCLUSION
Comorbidities are very common in biomass smoke-induced COPD. 20.83% (n=60) patients exposed to biomass smoke had one or the other comorbidity. The prevalence of CAD, HTN, HF and DM were more common in smoker COPD with significantly more prevalence of HTN (15.32% versus 3.4%), p < 0.0001) and CAD (13.86% versus 3.81%, p < 0.0001) in smokers. Further large scale studies are mandated to establish a causal relationship between biomass smoke exposure and these comorbidities.

REFERENCES
