

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

Dev Singh Jangpangi¹, Anubhuti Singh², Jagdish Rawat³, Tulsi Adhikari⁴

¹Assistant Professor, Department of Respiratory Medicine, Sri Guru Ram Rai Institute of Medical and Health Sciences, Dehradun, Uttarakhand.

²Assistant Professor, Department of Respiratory Medicine, Sri Guru Ram Rai Institute of Medical and Health Sciences, Dehradun, Uttarakhand.

³Professor, Department of Respiratory Medicine, Sri Guru Ram Rai Institute of Medical and Health Sciences, Dehradun, Uttarakhand.

⁴Scientist E, ICMR-National Institute of Medical Statistics, New Delhi.

ABSTRACT

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.

HOW TO CITE THIS ARTICLE: Jangpangi DS, Singh A, Rawat J, et al. 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. J. Evolution Med. Dent. Sci. 2018;7(16):1945-1948, DOI: 10.14260/jemds/2018/438

BACKGROUND

COPD is one of the major causes of morbidity and mortality worldwide. The burden of COPD in India is estimated to be around 14.84 million according to the 'Indian Study of Asthma Respiratory Symptoms and Chronic Bronchitis (INSEARCH)' conducted across 23 centres in India.¹ The most common risk factor for COPD is tobacco smoking. Another risk factor, which has gained prominence is emission from burning of biomass fuel.^{2,3}

This includes animal dung, wood, charcoal and crop residues used as a cooking fuel, especially in rural India. Females are predominantly affected, as they are involved in cooking indoors. There is 2.4 times more risk of developing COPD when females are exposed to biomass fuel as compared to other fuels.⁴ Also, those residing in cold climate and hilly terrains have greater exposure to fumes due to poor ventilation.⁵ Although, the major portion of COPD research has been done on tobacco smoke-induced COPD patients, the estimated number of smokers in the world (1.1 billion) is much lower than the number of people exposed to biomass smoke (3 billion).⁶ Exposure to biomass fuel contributes to around 4% of the global health burden.⁷

Biomass fuel provides 75% of domestic energy source in rural India. Incomplete combustion of biomass results in emission of harmful particulate matter, carbon monoxide, aromatic hydrocarbons, formaldehyde and sulfur dioxide.⁸ This inhaled smoke causes airway inflammation, airway injury and persistent bronchial hyper-responsiveness associated with goblet cell metaplasia and excessive mucus secretion.⁹ Among the various biomass fuels, burning of wood

'Financial or Other Competing Interest': None.
Submission 02-02-2018, Peer Review 01-04-2018,
Acceptance 07-04-2018, Published 16-04-2018.

Corresponding Author:

Dr. Anubhuti Singh,

Assistant Professor,

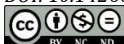
Sri Guru Ram Rai Institute of

Medical and Health Sciences,

Patel Nagar, Dehradun, Uttarakhand.

E-mail: anubhutikgm27@gmail.com

DOI: 10.14260/jemds/2018/438



generates higher concentration of PM¹⁰ (Respirable particulate matter with diameter $\leq 10 \mu\text{m}$) than charcoal.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.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^[1]- a diagnosis of COPD established according to GOLD guidelines.^[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^[1] co-existing respiratory illnesses such as pulmonary or pleural tuberculosis (TB), interstitial lung disease (ILD) or bronchiectasis.^[2] Patients with unstable coronary artery disease or arrhythmias. The spirometric parameters recorded for the purpose of the study was post bronchodilator FEV₁%, 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]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 \pm 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) FEV₁ %. 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 $22.3 \pm 3.99 \text{ kg/m}^2$, while that for biomass smoke-induced COPD was $23.08 \pm 5.65 \text{ kg/m}^2$ and this difference was also statistically significant ($p = 0.042$). There was no significant difference between mean post BD FEV₁ % among the two groups.

Variables	Smoking Induced COPD (n=411; 58.79%)	Biomass Fuel-Induced COPD (n=288; 41.20%)	P value
	Mean \pm SD	Mean \pm SD	
Age (Years)	62.6 \pm 11.0	59.2 \pm 10.98	<0.001
BMI (kg/m ²)	22.3 \pm 3.99	23.08 \pm 5.65	0.042
Post BD FEV ₁ %	46.0 \pm 14.08	47.51 \pm 14.5	0.169

Table 1. Characteristics of Tobacco Smoke-Induced and Biomass Fuel-Induced COPD

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.

Comorbidity	Tobacco Smoke-Induced COPD	Biomass Smoke-Induced COPD	P value
	%	%	
Systemic Hypertension	15.32	3.47	<0.0001
Coronary Artery Disease	13.86	3.81	<0.0001
Diabetes Mellitus	3.64	2.77	0.55
Heart Failure	6.32	5.55	0.70
Bronchogenic Carcinoma	2.18	2.43	0.86
Anaemia	1.70	2.08	0.70
Obstructive Sleep Apnoea	0.48	0.69	0.73

Table 2. Prevalence of Comorbidities among Tobacco Smoke-Induced and Biomass Fuel Smoke-Induced COPD

DISCUSSION

It has been documented that an average Indian woman has 60,000 hours of exposure to smoke from biomass fuel in her lifetime.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.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.¹³ Biomass fuel causes goblet cell metaplasia, increased mucus production and neutrophilic infiltration of bronchial wall.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 FEV₁ and different distribution of phenotypes in

comparison to smoking induced COPD.¹⁵ 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.¹⁶ 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.¹⁷

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.¹⁵ 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.¹⁶ Rivera reported that intimal thickening of pulmonary arterioles causing pulmonary hypertension is more common in patients exposed to biomass smoke than tobacco smoke.¹⁸

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.¹⁹ 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 3.47%, $p < 0.0001$) 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

- [1] Jindal SK, Aggarwal AN, Gupta D, et al. Indian study on epidemiology of asthma, respiratory symptoms and chronic bronchitis in adults (INSEARCH). *Int J Tuberc Lung Dis* 2012;16(9):1270-7.
- [2] Salvi S, Barnes PJ. Is exposure to biomass smoke the biggest risk factor for COPD globally? *Chest* 2010;138(1):3-6.
- [3] Torres-Duque C, Maldonado D, Perez-Padilla R, et al. Biomass fuel and respiratory disease. A review of the evidence. *Proc Am Thorac Soc* 2008;5(4):577-90.
- [4] Po JY, FitzGerald JM, Carlsten C. Respiratory diseases associated with solid biomass fuel exposure in rural women and children: systematic review and meta-analysis. *Thorax* 2011;66(3):232-9.
- [5] Hu G, Zhou Y, Tian J, et al. Risk of COPD from exposure to biomass smoke: a metaanalysis. *Chest* 2010;138(1):20-31.
- [6] Salvi SS, Barnes PJ. Chronic obstructive pulmonary disease in non-smokers. *Lancet* 2009;374(9691):733-43.
- [7] Bruce N, Perez-Padilla R, Albalak R. Indoor air pollution in developing countries: a major environmental and public health challenge. *Bull World Health Organ* 2000;78(9):1078-92.
- [8] Prasad R, Singh A, Garg R, et al. Biomass fuel exposure and respiratory diseases in India. *Biosci Trends* 2012;6(5):219-28.
- [9] Ezzati M, Kammen DM. The health impacts of exposure to indoor air pollution from solid fuels in developing countries: knowledge, gaps and data needs. *Environ Health Perspect* 2002;110(11):1057-68.
- [10] Dal Negro RW, Bonadiman L, Turco P. Prevalence of different comorbidities in COPD patients by gender and GOLD stage. *Multidiscip Respir Med* 2015;10(1):24.
- [11] Gaikwad DR. COPD in non smoker women with biomass fuel exposure (Chulha)-a review article. 2017;2(4).
- [12] Behera D, Jindal SK. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* 1991;100(2):385-8.
- [13] Mahesh PA, Jayaraj BS, Prabhakar JK, et al. Identification of a threshold for biomass exposure index for chronic bronchitis in rural women of Mysore district, Karnataka, India. *Indian J Med Res* 2013;137(1):87-94.
- [14] Mattson JD, Haus BM, Desai B, et al. Enhanced acute response in an experimental exposure model to biomass smoke inhalation in COPD. *Exp Lung Res* 2008;34(10):631-62.
- [15] Golpe R, Martin-Robles I, Sanjuan-Lopez P, et al. Prevalence of major co-morbidities in COPD caused by biomass smoke or tobacco. *Respiration* 2017;94(1):38-44.
- [16] Cheng LL, Liu YY, Su ZQ, et al. Clinical characteristics of tobacco smoke induced versus biomass fuel-induced COPD. *J Transl Int Med* 2015;3(3):126-9.

- [17] Camp PG, Ramirez-Venegas A, Sansores RH, et al. COPD phenotypes in biomass smoke- versus tobacco smoke-exposed Mexican women. *Eur Respir J* 2014;43(3):725-34.
- [18] Rivera RM, Cosio MG, Ghezzi H, et al. Comparison of lung morphology in COPD secondary to cigarette and biomass smoke. *Int J Tuberc Lung Dis* 2008;12(8): 972-7.
- [19] Solleiro-Villaricencio H, Quintana-Carrillo R, Falfan-Valencia R, et al. COPD induced by exposure to biomass smoke is associated with a Th2 cytokine production profile. *Clin Immunol* 2015;161(2):150-5.