International Journal of Medical Research Professionals
P-ISSN: 2454-6356; E-ISSN: 2454-6364
DOI: 10.21276/ijmrp

IJMRP

Original Research Article.

Pulmonary Hypertension in Patients with COPD Due to Biomass Smoke and Tobacco Smoke

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ABSTRACT

Introduction: Pulmonary hypertension (PH) is a common and well-established complication of chronic obstructive pulmonary disease (COPD). Its presence is associated with decreased survival. This study was designed to investigate the PH frequency and its relations in hospitalized tobacco and biomass related COPD patients.

Methodology: The study population in this retrospective study of 320 patients was conducted in COPD patients who had history of smoking or biomass fuel exposure and who had undergone echo-cardiography for evaluation of pulmonary hypertension. The duration of study was over a period of two year.

Result: The result of this study, patients exposed with biomass smoke for more than 10 years had increased incidence of pulmonary hypertension when compared to patients exposed to tobacco smoke for more than 10 years.

Conclusion: This study concludes that, suggest implementing strategies like medication of stove design, switching over to other high-efficiency & low-emission fuels for cooking to reduce exposure risk.

Keywords: COPD, Pulmonary Hypertension, Biomass Smoke, Tobacco Smoke.

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Article History:

Received: 12-04-2021, **Revised:** 06-05-2021, **Accepted:** 27-05-2021

Access this article online	
Website: www.ijmrp.com	Quick Response code
DOI: 10.21276/ijmrp.2021.7.3.005	

INTRODUCTION

It is well known that chronic obstructive pulmonary disease (COPD) is a major cause of chronic morbidity and mortality all over the world. According to WHO, It is also the 3rd leading cause of death in the world and further increases its prevalence and mortality can be predicted in the near future.[1,2]

Pulmonary hypertension (PH) is a major and well established complication of COPD.[3] It's presence is related with decreased survival.[4,5]

Tobacco smoking (TS) is significantly responsible for more than 80 % of the incidence of COPD.[6] COPD is also found in rural areas of developing countries. It is affecting mostly non-smoking women with exposure to biomass smoke (BS) during cooking. Biological fuels that produce heat are called biomass. It is observed that more than 90 % ofthe rural population in developing countries uses biological fuels.[7]

The present study is a retrospective study. This study is designed to investigate the Pulmonary Hypertension (PH) frequency and its relations in tobacco and biomass related COPD. Biomass smoke (BS) is composed of a relatively equal mixture of gases and particles. It can penetrate deeply into the lung. It produces a[8,9] variety of morphologic and biochemical changes. The association between Biomass smoke exposure, and Pulmonary hypertension and cor-pulmonale (CP) has long been established. Biomass smoke (BS) is composed of a relatively equal mixture of gases and particles. The routine investigation of Pulmonary hypertension is difficult in COPD patients due to request of right heart catheterization. The estimation about the prevalence of PH in patients with COPD vary widely. The prevalence of Pulmonary hypertension in COPD is mystified by several limiting factors. It is considered that Hypoxia is the major pathogenic mechanism of pulmonary[10] hypertension in COPD. It is suggested by histopathologic findings that the morphologic changes in the pulmonary arteries are initiated by the toxic effects of tobacco and biomass smoke. It progress in parallel with the parenchyma changes of COPD.

MATERIALS & METHODS

Study Population: The study population in this retrospective study of 320 patients was conducted in COPD patients who had history of smoking or biomass fuel exposure and who had undergone echo-cardiography for evaluation of pulmonary hypertension.

Study Duration: The duration of study was over a period of two year.

Study Area: This study was conducted in Department of Respiratory Medicine in Pacific Medical College Udaipur, Rajasthan.

Data Collection: Diagnosis of COPD was performed by assessment of functional criteria of chronic and irreversible air flow obstruction (forced expiratory volume in one second (FEV1)/(forced vital capacity) < 70 %, FEV1 < 80 % predicted) and without asthma as assessed by clinical history and response to bronchodilators (change <12% in FEV1 following 400 µg of inhaled salbutamol). Echocardiography was performed by our cardiologist by the using a Vivid 3 instrument (General Electric, US) and by utilizing a 2 MHz probe. Right ventricular systolic pressure (RVSP) can be estimated by measuring the TR jet maximum velocity by continuous wave (CW) spectral Doppler. If there is no significant stenosis at the right ventricular outflow tract, or the pulmonic valve, the RVSP is equivalent to the systolic pulmonary artery pressure (SPAP). Elevated right ventricular systolic pressure (RVSP >25mmhg) calculated through echocardiography was taken as having pulmonary hypertension. Patients are graded according to the RVSP Values into mild (25-40), moderate (40-55) and severe (>55) PH Patients were further categorized into total tobacco smokers, beedi smokers, cigarette smokers, total biomass exposure, biomass exposure <10 years duration, biomass exposure > 10 years duration and the RVSP values are plotted accordingly against each individual category.

Data Analysis: Data were analysed by using Microsoft Excel.

Table:1 Distribution of cases according to exposure with elevated RVSP

Exposure	Total cases	Elevated RVSP	Percentage
Tobacco smoke	204	112	55%
Biomass exposure	116	68	58%

Table:2 Distribution of cases according to categorization

	Beedi	Cigarette	Biomass
Cases	68	136	116
PAH	52	60	68

Table:3 Distribution of cases according to exposure & severe pulmonary hypertension

Exposure	Cases	Severe PH	Percentage
Beedi	68	4	5%
Cigarette	136	24	17.6%
Biomass<=10year	32	0	0
Biomass>=10year	12	12	14.3

RESULTS

In our study, 112 among the 204 cases of tobacco smoke exposure (55%) had elevated RVSP, while 68 cases out of 116 cases of biomass fuel exposure (58%) had elevated RVSP. Patients exposed to cigarette smoke had less prevalence of pulmonary hypertension as compared to other types of smoking. Patients exposed to biomass smoke for more than 10 years had increased incidence of pulmonary hypertension when compared to patients exposed to tobacco smoke for more than 10 years.

DISCUSSION

A recent meta-analysis reviewed risk of COPD from exposure to BS. It concluded that BS exposure is a clear risk factor for COPD.[11] BS related COPD cases' clinical characteristics, quality of life, and mortality rate were similar in degree to that of tobacco smokers.[12] In this study we observed that the biomass related COPD cases had more frequent PH than cigarette smoking related COPD cases. The relationship between BS and PH and corpulmonale (CP) has long been established.[13-15] A study was conducted in females with CP in Delhi, India. It reported that those females with CP had biomass smoke exposure history. They found that PH and CP development might have a correlation with biomass smoke exposure.

In another study, it has been found that women who were exposed to wood smoke had chronic pulmonary disease and a significantly high PH.[16] A study compared the COPD cases caused by BS exposure and tobacco smoking. Results revealed that vascular changes were prominent in both groups but were more severe in the biomass smoke exposed group. These findings could explain why PH and CP in women exposed to biomass smoke is high.[17] It has been previously reported that prevalence of PH was higher among BS exposed females than non-exposed females in a healthy cohort (48 % vs 12 %, $p<0.05$). The Odds Ratio for PH development with BS exposure was established as 6 ($p<0.001$).[18] Independent predictors of PH were found in moderate level, severe level, and very severe level COPD cases as follow; gender, FEV1 %, FVC %, PaO2, PaCO2, and PaO2, PaCO2 and gender, FEV1%, FVC % respectively.

Previous studies examining the relationship between spirometry and PH reported similar results.[19-21] Though some studies reported no associations between FEV1 and sPAP.[10,22] It is reported by Sandoval et al that there is a correlation between pulmonary artery pressure (PAP) and PaO2 in wood smoke exposed women, but there was not any correlation regarding other factors such as FEV1 %, FVC %, and PaCO2. Similar findings

were found by Sim et al.[23] A study examines the relation between PH and inflammation in COPD and found that PaO2 and C-reactive protein were independent variables for PH in COPD cases.[24] These contradictory findings may be due to the differences in the severity of COPD between cohorts.

The finding of this study that biomass smoke has a greater risk for PH in women may suggest a predisposition to PH in females. It is seen that age is an independent predictor of PH in COPD. It is suggested that this increasing may be relating to decreasing of FEV1 % and FVC %. In this study, however, age was not an independent factor in any COPD level. Though the prevalence of PH was increased accordingly COPD level. This finding is different from previous studies.

CONCLUSION

This study concludes that, pulmonary hypertension frequency is almost equal with tobacco smoke and biomass fuel exposure. Frequency of pulmonary hypertension is more significant in biomass smoke exposure for more than 10years duration. Therefore, biomass exposure should always be considered as an important etiological agent for COPD. We suggest implementing strategies like medication of stove design, switching over to other high-efficiency & low-emission fuels for cooking to reduce exposure risk.

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Source of Support: Nil. **Conflict of Interest:** None Declared.

Cite this article as: Sunil Kumar, Manish Advani, Jyoti Kumari, Sheena Taneja. Pulmonary Hypertension in Patients with COPD Due to Biomass Smoke and Tobacco Smoke. Int J Med Res Prof. 2021 May; 7(3): 16-18. DOI:10.21276/ijmrp.2021.7.3.005