ORIGINAL RESEARCH PAPER

Volume-8 | Issue-3 | March-2019 | PRINT ISSN No 2277 - 8179

INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

PULMONARY HYPERTENSION IN PATIENTS WITH COPD DUE TO BIOMASS SMOKE AND TOBACCO SMOKE

Pulmonary Medicine

Dr. Karthik Tipparapu PG, Department Of Pulmonary Medicine, Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences, Gannavaram, Andhrapradesh.

Dr. Swathi Rosa. K* PG, Department Of Pulmonary Medicine, Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences, Gannavaram, Andhrapradesh. *Corresponding Author

Dr. B. Bhanu Rekha Prof and HOD, Department Of Pulmonary Medicine, Dr. Pinnamaneni Siddhartha Institute Of Medical Sciences, Gannavaram, Andhrapradesh.

ABSTRACT

Aims&objective: To study the incidence&severity of pulmonary hypertension(PH) in tobacco and biomass related COPD patients.

Materials&Methods: This was a retrospective review of in-patients with COPD either with tobacco/biomass fuel exposure. Case-records of 160 patients admitted in department of pulmonary medicine during 2017-2018 were reviewed. Elevated right ventricular systolic pressure(RVSP >25) calculated through echocardiography was taken as pulmonary-hypertension.

Results&conclusion: In this study 56 outof 102 patients of tobacco exposure(55%) and 34 outof 58 patients of biomass fuel exposure(58%) had elevated RVSP. Patients exposed to cigarette smoke had less incidence of pulmonary-hypertension compared to beedi/chutta,but it was not statistically significant.

We conclude that occurrence of pulmonary-hypertension is almost equal with tobacco smoke and biomass fuel exposure,but severity to biomass-smoke for >10years had high incidence of pulmonary-hypertension(76%) when compared to tobacco-smoke for >10years(48.30%) and is significant (pvalue-0.00).

KEYWORDS

COPD, Tobacco, Biomass, RVSP

Introduction:

Chronic obstructive pulmonary disease (COPD) is a major cause of chronic morbidity and mortality throughout world. Pulmonary hypertension (PH) is a common and well established complication of COPD .[1] Its presence is associated with decreased survival.[2,3] It is predicted that half of the world population and more than 90 % of the rural population in developing countries uses biological fuels.[4]

Aims and objectives:

This is a retrospective study designed to investigate the PULMONARY HYPERTENSION (PH) frequency and its relations in tobacco and biomass related COPD.

Patients and methods:

Subjects were chosen from the patients presented to OPD AND IN-PATIENTS in the department of pulmonology of Dr.PSIMS & RF. Retrospective review of 160 patients was conducted on COPD patients who had history of smoking or biomass fuel exposure and who had undergone echo-cardiography for evaluation of pulmonary hypertension between the years of 2017and 2018. All patients considered were clinically stable. Patients with conditions including valvular abnormalities , left heart failure, pulmonary embolus, sleep apnea, obesity (BMI > 35) were excluded.

Diagnosis of COPD was performed by assessment of functional criteria of chronic and irreversible airflow 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[5] Patients were further categorized into total tobacco smokers,beedi/chutta smokers, cigarette smokers, total biomass exposures, biomass exposure<10 years duration, biomass exposure > 10 years duration and the RVSP values are plotted accordingly against each individual category.

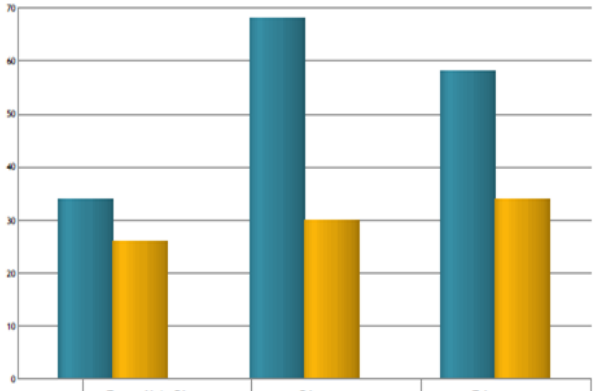
Observation:

In this study 56 out of 102 patients of tobacco smoke exposure(55%) had elevated RVSP 34 out of 58 patients of biomass fuel exposure(58%) had elevated RVSP.

EXPOSURE	CASES	PH	PERCENTAGE
TOBACCO SMOKE	102	56	55%
BIOMASS EXPOSURE	58	34	58%

p-0.641 ; chi square-5.26 –no significant relation.

Patients after categorizing into beedi/chutta, cigarette smokers and biomass exposure



	Beedi/ Chutta	Cigarette	Biomass
Cases	34	68	58
PAH	26	30	34
%	76.4%	44%	58.6%

p-0.228 : no significant relation.

Patients of all categories having severe PAH were isolated and their percentage is calculated

Patients exposed to cigarette smoke had less incidence of pulmonary

EXPOSURE	CASES	SEVERE PH	PERCENTAGE
BIDIE/CHUTTA	34	2	5%
CIGARETTE	68	12	17.60%
BIOMASS <= 10 YERAS	16	0	0%
BIOMASS >= 10 YERAS	42	6	14.30%

hypertension compared to other types of smoking, but it was not statistically significant.

Patients exposed to biomass smoke for more than 10 years had high incidence of pulmonary hypertension(76%) when compared to patients exposed to tobacco smoke for more than 10 years(48.30%) and is statistically significant (p value-0.00).

DISCUSSION:

Biomass smoke (BS) is composed of a relatively equal mixture of gases and particles and can penetrate deeply into the lung, producing a variety of morphologic and biochemical changes.[6,7]

The relationship between Biomass smoke exposure, and Pulmonary hypertension and cor-pulmonale (CP) has long been established.[8] Biomass smoke (BS) is composed of a relatively equal mixture of gases and particles and can penetrate deeply into the lung, producing a variety of morphologic and biochemical changes.[6,7]

The relationship between Biomass smoke exposure, and Pulmonary hypertension and cor-pulmonale (CP) has long been established .[8] The routine investigation of Pulmonary hypertension is difficult in all COPD patients due to request of right heart catheterization. Estimates of the prevalence of PH in patients with COPD vary widely.

The literature on the prevalence of Pulmonary hypertension in COPD is confounded by several limiting factors. Hypoxia has been classically considered to be the major pathogenic mechanism of pulmonary hypertension in COPD .[9] Early histopathologic findings suggest that the morphologic changes in the pulmonary arteries are initiated by the toxic effects of tobacco and biomass smoke and progress in parallel with the parenchymal changes of COPD .[10]

The results were simillar to the study conduted by Department of Pulmonary and Critical Care, Yuzuncu Yil University Medical Faculty, Turkey in 2015.

CONCLUSION:

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 modification of stove design,switching over to other high-efficiency & low-emission fuels for cooking to reduce exposure risk .

REFERENCES:

1. Burrows, B., Kettel, L. J., Niden, A. H., Rabinowitz, M., & Diener, C. F. (1972). Patterns of cardiovascular dysfunction in chronic obstructive lung disease. New England Journal of Medicine, 286(17), 912-918.
2. Weitzenblum, E., Hirth, C., Ducolone, A., Mirhom, R., Rasaholinjanahary, J., & Ehrhart, M. (1981). Prognostic value of pulmonary artery pressure in chronic obstructive pulmonary disease. Thorax, 36(10), 752-758.
3. Traver, G. A., Cline, M. G., & Burrows, B. (1979). Predictors of mortality in chronic obstructive pulmonary disease: a 15-year follow-up study. American review of respiratory disease, 119(6), 895-902.
4. WHO/UNICEF. Integrated management of childhood illness. Geneva, World Health Organization, 1997 (unpublished document WHO/CHD97.3.A-G).
5. Chaouat, A., Naeije, R., & Weitzenblum, E. (2008). Pulmonary hypertension in COPD. European Respiratory Journal, 32(5), 1371-1385.
6. Zelikoff, J. T., Chen, L. C., Cohen, M. D., & Schlesinger, R. B. (2002). The toxicology of inhaled woodsmoke J Toxicol Environ Health B Crit Rev 5 (3): 269–282. Find this article online.
7. Montano, M., Beccerril, C., Ruiz, V., Ramos, C., Sansores, R. H., & González-Avila, G. (2004). Matrix metalloproteinases activity in COPD associated with wood smoke. Chest, 125(2), 466-472.
8. de Koning, H. W., Smith, K. R., & Last, J. M. (1985). Biomass fuel combustion and health. Bulletin of the World Health Organization, 63(1), 11.
9. Higham, M. A., Dawson, D., Joshi, J., Nihoyannopoulos, P., & Morrell, N. W. (2001). Utility of echocardiography in assessment of pulmonary hypertension secondary to COPD. European Respiratory Journal, 17(3), 350-355.
10. Hale, K. A., Niewoehner, D. E., & Cosio, M. G. (1980). Morphologic changes in the muscular pulmonary arteries: relationship to cigarette smoking, airway disease, and emphysema. American Review of Respiratory Disease, 122(2), 273-278.