Original Research Paper

Volume - 12 | Issue - 09 | September - 2022 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Respiratory Medicine

COPD: NOT JUST SMOKER' S ILLNESS!

Dr. Pragati Mikkilineni	MBBS MD, Pulmonary Medicine Assistant Professor, NIMRA INSTITUTE OF MEDICAL SCIENCES, Ibrahimpatnam, Vijayawada, AP- 521456
Dr. Nalini Ghanate	MBBS MD DNB DAA, Assistant Professor, Pulmonary Medicine, Government General TB and Chest Hospital, allied to Osmania Medical College, Hyderabad 500018
Dr. Rajesh Palvai	MBBS, MD Assistant Professor, Pulmonary Medicine, Government General TB and Chest Hospital, allied to Osmania Medical College, Hyderabad 500018
Dr. Sameena Pinjari*	MBBS, MD, Pulmonary Medicine, Senior Resident Government General TB and Chest Hospital, allied to Osmania Medical College, Hyderabad 500018 *Corresponding Author
Dr A Satya Prasad	MBBS, MD HOD and Professor, Department of Pulmonary Medicine, Mamatha Medical College, Khammam-Telangana

ABSTRACT **Background:** Although tobacco smoking is the best-studied COPD risk factor, it is not the only one and there is consistent evidence from epidemiologic studies that non-smokers may also develop chronic airflow limitation. Although several longitudinal studies of COPD have followed groups and populations for up to 20 years, none has monitored the progression of the disease through its entire course, or has included the pre-and perinatal periods which may be important in shaping an individual's future COPD risk. Thus, current understanding of risk factors for COPD is in many respects still incomplete.1. Early diagnosis and treatment, is needed to slow the progression of symptoms and reduce flare-ups.2 **Aim:** To study the clinical profile of COPD in non-smokers, also identify the other risk factors (other than smoking) of COPD. **Methods:** This is institution based descriptive study conducted that included 50 patients with COPD who had post-bronchodilator FEV1/FVC < 0.7 who are non-smokers. Patients with smoking history, bronchial asthma, Pulmonary tuberculosis (present or past), interstitial lung disease, heart failure were excluded. **Results:** There were 38 females, 12 males in this study which had total of 50 patients. 84% of patients had exposure to biomass fuel, 100% of the patients had exposure to environmental tobacco which was statistically significant (p=0.001). 34% of patients had occupational exposure, 16% of patients had exposure to air pollution which were not statistically significant. This was supported by PFT, pre-and post FEV1 change which was statistically significant (p=0.001) **Conclusion:** Biomass fuel usage and environmental tobacco smoke exposure were the most common risk factors. In this study occupational exposure and air pollution were not associated with increased risk of COPD. Pulmonary function test showed no significant change in pre- and post-bronchodilator FEV1/FVC.

KEYWORDS : COPD; non-smokers, biomass fuel exposure, air pollution, spirometry, FEV1/FVC.

INTRODUCTION:

Chronic obstructive pulmonary disease (COPD) is the third leading cause of death worldwide, causing 3.23 million deaths in 2019.Nearly 90% of COPD deaths in those under 70 years of age occur in low- and middle-income countries (LMIC). In 2019, 212.3 million prevalent cases of COPD were reported globally, with COPD accounting for 3.3 million deaths and 74.4 million DALYs [3]. Globally, COPD burden is projected to increase in coming decades because of continued exposure to COPD risk factors and aging of the population. Prevalence and morbidity data greatly underestimate the total burden of COPD because the disease is usually not diagnosed until it is clinically apparent and moderately advanced.[4]

Exposure to Environmental tobacco smoke (ETS), which contains potent respiratory irritants, may lead to chronic airway inflammation and obstruction. Although ETS exposure appears to cause asthma in children and adults, its role in causing COPD has received less attention in epidemiologic studies. A body of literature now supports an association between ETS exposure and the development of COPD independent of personal cigarette smoking.[5]

In developing countries, a significant proportion of COPD cases occurs among never-smokers, especially in women cooking with open fire stoves. The fuel used in these stoves is collectively known as biomass, which includes wood, animal dung, and crop residues. World Health Organization estimates that 35% of COPD in low- and medium-income countries is from indoor smoke from solid fuels.[6]

COPD does not have a clinical subcategory that is clearly identified as occupational, largely because the condition develops slowly and, given that the airway obstruction is chronic, does not reverse when exposure is discontinued. Consequently, a diagnosis of "occupational COPD" is rarely made by clinicians; this situation is in sharp contrast to occupational asthma, which is more frequently recognized. Perhaps the strongest evidence implicating occupational exposures in the pathogenesis of COPD comes from community- based studies [7,8,9,10.] Beatrix Groneberg-Kloft et al studied causes of chronic cough and concluded that next to the well-known air pollutants which also include particulate matter and sulphur dioxide, a number of other indoor and outdoor pollutants have been demonstrated to cause chronic cough and therefore, environmental factors have to be taken into account as potential initiators of adult cough.[11]

MATERIAL AND METHODS

Ethical approval

The study was approved by the Institutional Ethics Committee of Mamata Medical College, Telangana

Study setting and Patient selection

This is an Institutional based descriptive study conducted over a period of two years .Fifty patients who presented with history of cough, sputum, breathlessness or wheezing of more than 3 months duration to the medical outpatient or admitted in pulmonary medicine wards of Mamatha General Hospital were subjected to pre- and post-bronchodilator pulmonary function testing. Those patients whose post-bronchodilator FEV1/FVC was less than 0.7 were included in this study. Patients with history of smoking, Bronchial asthma, Pulmonary tuberculosis (present or past), Interstitial lung disease, Heart failure were excluded. Data was collected using a pretested proforma. Detailed history, physical examination, necessary investigations and consent were undertaken. Pulmonary function testing was done using Micro Lab [Micro Medical Ltd., U.K.] spirometer. Three satisfactory efforts were recorded and best effort was considered. Bronchodilatation was done using 200 µg of inhaled salbutamol using a metered dose inhaler and test was repeated after 15 min.

Data analysis

All the statistical methods were carried out through the SPSS for

Windows (version 16.0) and Minitab (version 11.0) for windows.

RESULTS

There were total of 50 patients in this study, 76% females and 24% males. Mean age of the studied patients was 43.9 ± 12.8 years. Majority of the patients were in the age group of 41-50 years. 42 patients (84%) were living in rural area.

Out of 50 patients, 42 patients (84%) gave history of biomass fuel usage and exposure. All 8 patients (16%) who did not have biomass fuel exposure were males. Of the 42 patients, 10 patients (20%) gave history of exposure <6 hours/day, 32 patients (64%) gave history of exposure >6 hours/day. Six patients (12%) gave duration of exposure for biomass fuel <10 years whereas 36 patients (72%) gave history of duration of exposure for biomass fuel for >10 years. Increased duration of exposure to biomass fuel was associated with increased risk of COPD. Thirty-three patients (66%) gave history of firewood usage and 9 patients (18%) gave history of firewood and cow dung usage.

Table 1: Showing risk factors for COPD in non-smokers

Risk factor exposure	Number of patients and percentage	p-value
1.Bio mass Exposure	42 (84%)	p< 0.001
2. More than 6 hours of exposure/day	32 (64%)	p< 0.001
3. More than 10 Years of exposure	36 (72%)	p< 0.001
4. Fire wood alone used as Fuel	33 (66%)	p< 0.0001
5. Firewood & Cowdung used as fuel	9 (18%)	p< 0.0001
6.Environmental tobacco exposure	50(100%)	p< 0.0001
7. greater than 3 hours of exposure	49 (98%)	p< 0.001
8. Air Pollution	08 (16%)	
9.Occupational exposure	17(34%)	

History of exposure to environmental tobacco smoke was present in all 50 patients (100%). Most of the patients, i.e., 49 patients (98%) gave history of environmental tobacco smoke exposure for >3 hours, only 1 patient (2%) gave history of exposure for <3 hours in a day. History of exposure to air pollution was present in 8 patients (16%) which was statistically not significant. In the present study, occupational exposure was 34% which was comparable to other studies.

Table 02: Showing Pulmonary Function Tests

	Pre-tests	Post test	P value
FEV1	1.5± 0.54	1.7± 0.5	0.001
FVC	2.9 ± 1.95	3.01± 1.3	0.796
FEV1/FVC	55.05 ± 15.9	59.4 ± 17.7	0.064
PEFR	2.76 ± 1.5	3.09 ± 1.5	0.032

Spirometry is the most robust test of airflow limitation in patients with COPD. A low FEV1with an FEV1/FVC ratio below the normal range is a diagnostic criterion for COPD [12]. On pulmonary function test, there was no significant change in pre-FVC (2.9±1.45) and post-FVC (3.01±1.3). Pre-FEV1 (1.5±0.54) and post-FEV1 (1.7±0.5) change was significant statistically.

DISCUSSION

In the past, most studies showed that COPD prevalence and mortality were greater among men than women but data from developed countries (Mannino DM et al [4]) show that the prevalence of the disease is now almost equal in men and women. Some studies have even suggested that women are more susceptible to the effects of tobacco smoke than men (Menezes AM et al [13], Larsson ML et al [14])

Environmental tobacco smoke exposure at home and/or at work was 100% in the present study, which is comparable with Berglund et al [15] and Mahesh et al. [16]study.

Approximately half of the world's population uses solid fuels for cooking; usage is even higher in rural areas (up to 80%). Particulate matter concentrations in these kitchens are very high, with average values in the range of milligrams per cubic meter and peak levels reaching 10–30 kg/m^3. [17] Many studies have identified biomass smoke as a primary risk factor for COPD in rural areas. Results from studies in India [18], China [19], Turkey[20], Mexico [21], Nepal [22.] and Pakistan [23] have shown that women using biomass fuel for cooking have increased prevalence of respiratory symptoms attributable to COPD and substantially greater decline in lung function than women who do not use these fuels.

The demonstration of an association between occupational exposures and COPD in epidemiological studies can be difficult because of several factors. First, COPD is multifactorial in etiology, with critical (and mostly unknown) host as well as non-occupational environmental determinants of risk. Second, unlike workers with pneumoconiosis, individuals with COPD due to occupational exposures cannot be distinguished from those with the disease due to other causes. Third, workforce studies are often limited to a "survivor" population because of inability to assess or monitor workers who leave their jobs, thereby under- estimating the chronic effects of occupational exposures.

In the present study, 84% subjects were from rural background, which was comparable to Goel S et al. study [24]. In the present study, pre and post bronchodilator FEV1/FVC was and the percentage change in pre and post bronchodilator was 7.3% in the present study which was comparable to Zhou et al. study. [25]

CONCLUSION

1. In this study of COPD in non-smokers, females were predominant.
2. Environmental tobacco smoke exposure and biomass fuel usage were significant risk factors.
3. According to this study, occupational exposure and air pollution were not significant risk factors.

Limitations

1. Sample size was small, i.e., only 50.
2. It was a hospital-based study. Only symptomatic patients who presented to hospital were studied.
3. Exact quantification of biomass fuel exposure, environmental tobacco smoke exposure, occupational exposure and air pollution was not done.

REFERENCES

1. GOLD guidelines 2022 updated.
2. World Health Report. Geneva 2022: World Health Organization -https:// www. who.int /news - room/ fact-sheets/detail/chronic-obstructive-pulmonary-disease-(copd)
3. Safiri S, Carson-Chahhoud K, Noori M, Nejadghaderi S A, Sullman M J M, Ahmadian Heris J et al. Burden of chronic obstructive pulmonary disease and its attributable risk factors in 204 countries and territories, 1990-2019: results from the Global Burden of Disease Study 2019 BMJ 2022; 378 :e069679 doi:10.1136/bmj-2021-069679
4. Raherison C, Girodet PO. Epidemiology of COPD. Eur Respir Rev 2009;18(114):213-21
5. Mark D Eisner, John Balmes, Patricia P Katz, et al. Lifetime environmental tobacco smoke exposure and the risk of chronic obstructive pulmonary disease; Environmental Health: A GlobalAccess Science Source 2005;4:7
6. David M Mannino. Looking beyond the cigarette in COPD. Chest 2008; 133:333-4.
7. Hnizdo F, Patricia Sullivan, Ki Moon Bang, et al. Association between COPD and Employment by industry and occupation in the US population: A study of data from the 3rdNHANES. American Journal of Epidemiology 2002; 156:738-46.
8. Tomas ML Eagan, Amund Gulsvik, Geir E Eide, et al. Occupational Airborne Exposure and the Incidence of Respiratory Symptoms and Asthma. Am J Respir Crit Care Med 2002;166:933-8.
9. Per S Bakke, Baste V, Hanoa R, et al. Prevalence of obstructive lung disease in a general population: relation to occupational title and exposure to some airborne agents. Thorax 1991;46:863-70.
10. Matheson MC, Benke G, Raven J, etal. Biological dust exposure in the workplace is a risk factor for COPD. Thorax 2005;60:645-51.
11. Beatrix Groneberg-Kloft, Thomas Kraus,Anke Van Mark, et al. Analysing the causes of chronic cough: relation to diesel exhaust, ozone, nitrogen oxides, sulphur oxides and other environmental factors. Journal of Occupational Medicine and Toxicology 2006,1:6.
12. Jindal SK. Spirometers. Lung India 1993;11(1&2):60-3.
13. Menezes AM, Perez-Padilla R, Jardim JR, et al. Chronic obstructive pulmonary disease in five Latin American cities (the PLATINO study): a prevalence study. Lancet 2005;366:1875-81.
14. Larsson ML, Loit HM, Meren M, et al. Passive smoking and respiratory symptoms in the FINESS Study. Eur Respir J 2003;21:672-6.
15. David J Berglund, David E Abbey, Michael D Lebowitz, et al. Respiratory Symptoms and Pulmonary Function in an Elderly Nonsmoking Population. Chest 1999;115:49-59.
16. Mahesh PA, Jayaraj BS, Prahalad T,et al. Validation of a structured questionnaire for COPD and prevalence of COPD in rural area of Mysore; a pilot study. Lung India 2009 Jul-Sep;26(3).
17. Padmavati S, Pathak SN. Chronic CorPulmonale in Delhi: A Study of 127 Cases. Circulation 1959;20:343-52
18. Behera D, Jindal SK. Respiratory Symptoms in Indian Women using Domestic Cooking Fuels. Chest 1991;100:385-8.
19. Shengming Liu, Yumin Zhou, Xiaoping Wang, et al. Biomass fuels are the probable risk factor for chronic obstructive pulmonary disease in rural South China. Thorax 2007;62:889-97.
20. Kiraz K, Kart L, Demir R, et al. Chronic pulmonary disease in rural women exposed to biomass fumes. Clin Invest Med2003; 26:243–48
21. Justino Regalando, Rogelio Perez Padilla, Raul Sansores, et al. The Effect of Biomass Burning on Respiratory Symptoms and Lung Function in Rural Mexican Women. Am J RespirCrit Care Med 2006;174:901-5.
22. Pandey MR . Domestic smoke pollution and chronic bronchitis in a rural community of

the Hill Region of Nepal. Thorax 1984;39:337-9.
23. Tasleem Akhthar, Zahoor Ullah, Mir Hassan Khan, et al. Chronic bronchitis in women using solid biomass fuel in rural Peshawar, Pakistan. Chest 2007;132;1472-5.
24. Sonu Goel, Gupta BP, Kashyap S, Bhardwaj AK. Epidemiological Aspects of Chronic Bronchitis in Shimla Hills.
25. ZhouY,Wang C et al .COPD in Chinese nonsmokers. Eur Respir J 2009; 33: 509–518