Section: Respiratory Medicine

Original Research Article

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

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**ABSTRACT**

**Background:** Biomass fuel, a heat source derived from biological materials, is relied upon by over 90% of rural populations in developing countries. Alarmingly, an estimated 3 billion people worldwide are exposed to biomass smoke, surpassing the 1.01 billion tobacco smokers. This suggests that biomass smoke exposure may be the leading risk factor for Chronic Obstructive Pulmonary Disease (COPD) globally.
**Material and Methods:** This study was conducted at the Department of Respiratory Medicine, Dr. S.N. Medical College in Jodhpur, Rajasthan. Over a two-year period, 320 participants were included in the study.
**Results:** Among the 320 COPD patients, 63.7% reported exposure to tobacco smoke, while 36.3% were exposed to biomass smoke. Notably, the study revealed a strong correlation between prolonged exposure to biomass and cigarette smoke and the development of severe Pulmonary Hypertension (PAH).
**Conclusion:** This study underscores the critical role of biomass exposure as a causative agent in the development of COPD. Recognition of biomass exposure as a key risk factor is essential for developing effective prevention and management strategies.
**Key Words:** COPD, biomass exposure, tobacco smoke.

## INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a leading cause of mortality worldwide, with pulmonary hypertension (PAH) being a common complication that decreases survival rates.[1,2] In industrialized countries, tobacco smoking (TS) is responsible for over 80% of COPD cases.[3] However, in rural areas of developing countries, biomass smoke exposure during cooking is a significant risk factor, particularly affecting non-smoking women.

Biomass fuel, which generates heat from biological materials, is used by over 90% of the rural population in developing countries.[4] Globally, approximately 3 billion people are exposed to biomass smoke, compared to 1.01 billion tobacco smokers. This suggests that biomass smoke exposure may be the most significant risk factor for COPD worldwide.

COPD is a major cause of mortality in developing countries, primarily due to environmental pollution, biomass or tobacco smoke, and industrialization. In India, the mortality rate is 102.3 per 100,000, with a global mortality rate of 6,740,000 per day.[5]

Studies have shown an increasing trend of COPD and its complications, including PAH, left ventricular failure, and right ventricular failure.[6,7] These conditions significantly impact daily activities and quality of life. In rural areas, the incidence of COPD is higher among those exposed to biomass smoke than tobacco smoke.

This study aims to detect PAH in COPD patients with a history of exposure to biomass smoke and tobacco smoke using investigations.

## MATERIALS AND METHODS

**Study Area:** This study was conducted in Department of Respiratory Medicine, Dr S.N. Medical College Jodhpur, Rajasthan.

**Study Duration:** The Duration of study was over a period of two years.

**Study population:** The population were included in this study was 320.

**Data collection:** Diagnosis and Grading of COPD and Pulmonary Hypertension

COPD diagnosis was based on functional criteria, including:

- Chronic and irreversible airflow obstruction
- Forced expiratory volume in one second (FEV1)/forced vital capacity (FVC) ratio < 70%
- FEV1 < 80% predicted
- Absence of asthma, assessed through clinical history and response to bronchodilators (change > 200 mmHg)

**Pulmonary hypertension (PAH)** is defined as a mean pulmonary artery pressure(mPAP)> 20 mmHg at rest, measured by right heart catheterization.

PAH is considered **Severe** if mPAP is ≥ 35 mmHg **or** the mPAP is ≥ 20 mmHg with an elevated right atrial pressure **and/or** cardiac index is <2 L/min/m

**PAH definition according to 6thWSPAH.[8]**

| Definition | Characteristics | Clinical groups (WHO Group) |
|---|---|---|
| Pre-capillary PAH | mPAP > 20 mmHg, PAWP ≤ 15 mmHg, PVR ≥ 3 WU | 1, 3, 4, 5 |
| Isolated post-capillary PAH | mPAP > 20 mmHg, PAWP > 15 mmHg, PVR < 3 WU | 2, 5 |
| Combined pre- and post-capillary PAH (CpcPAH) | mPAP > 20 mmHg, PAWP > 15 mmHg, PVR > 3 WU | 2, 5 |

Abbreviations:
- mPAP: Mean pulmonary arterial pressure
- PAWP: Pulmonary arterial wedge pressure
- PVR: Pulmonary vascular resistance
- WU: Wood unit

**Categorization of Patients**

Patients were categorized into:
- Total tobacco smokers
- Beedi/Chutta smokers
- Cigarette smokers
- Total biomass exposure
- Biomass exposure ≥ 10 years

**Data Analysis:** Data were analysed by using Microsoft excel.

## RESULTS

A study of 320 COPD patients revealed that 63.7% were exposed to tobacco smoke, while 36.3% were exposed to biomass. This indicates that tobacco smoke is the primary risk factor for COPD, but biomass exposure is also a significant concern that cannot be overlooked.A total of 180 pulmonary arterial hypertension cases were identified, with 112 cases attributed to tobacco smoke exposure and 68 cases linked to biomass smoke exposure.

Among the 204 tobacco smoke-related cases, a significant proportion developed pulmonary arterial hypertension. Specifically, 52 out of 68 Beedi/Chutta smokers and 60 out of 136 cigarette smokers were affected, totalling 112 cases. This highlights the strong link between tobacco smoke exposure and the development of pulmonary arterial hypertension.A significant association was found between exposure to COPD risk factors and the development of severe pulmonary hypertension (PAH), with a P value of 0.00. Notably, cigarette smokers were more likely to develop severe PAH, with 24 out of 136 cases affected. Beedi/Chutta smokers also showed a significant risk, with 4 out of 68 cases developing severe PAH. In contrast, biomass exposure for 10 years or less did not result in any severe PAH cases. However, prolonged biomass exposure of 10 years or more increased the risk, with 12 out of 84 cases developing severe PAH. Overall, the study found a strong correlation between long-term exposure to biomass and cigarette smoke and the development of severe PAH.

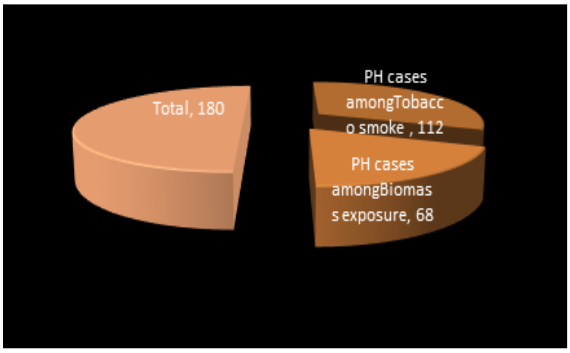


**Figure 1: Chart showing pulmonary hypertension cases among the biomass exposure and Tobacco smoke exposure cases**

**Table 1: Distribution of cases according to Exposure**

| Exposure | Number of cases | Percentage |
|---|---|---|
| Tobacco smoke | 204 | 63.7% |
| Biomass exposure | 116 | 36.3% |
| Total | 320 | 100% |

**Table 2: Distribution of cases according to Beedi/Chutta and Cigarette cases among the Tobacco smoke cases**

| Tobacco smoke cases | Number of cases | Pulmonary Arterial hypertension |
|---|---|---|
| Beedi/Chutta | 68 | 52 |
| Cigarette | 136 | 60 |
| Total | 204 | 112 |

**Table 3: Distribution of cases according to severe pulmonary hypertension cases among the all cases**

| Exposure | Cases | Severe PAH | |
|---|---|---|---|
| Beedi/Chutta | 68 | 4 | P value 0.00 |
| Cigarette | 136 | 24 | |
| Biomass Years ≤10 | 32 | 0 | |
| Biomass Years ≥ 10 | 84 | 12 | |
| Total | 320 | 40 | |

## DISCUSSION

Biomass smoke (BS) is a harmful mixture of gases and particles that can penetrate deeply into the lungs, causing various morPAHological and biochemical changes.[9,10] The link between BS exposure and pulmonary hypertension (PAH) and corpulmonale (CP) has been well-established.[11]Investigating PAH in COPD patients is challenging due to the need for right heart catheterization. Estimates of PAH prevalence in COPD patients vary widely, and the literature is limited by several factors. Hypoxia is considered a primary mechanism for PAH development in COPD.[12]Histopathologic findings suggest that toxic effects of tobacco and biomass smoke initiate morPAHological changes in pulmonary arteries, progressing in parallel with COPD parenchymal changes.[13] PAH presence in COPD patients is associated with high mortality. PAH limits exercise performance in tests like the 6MWT and cardiopulmonary exercise test. While classical teaching attributes PAH development in COPD due to hypoxia induced pulmonary vasoconstriction and vascular bed destruction, multiple factors are now recognized, including endothelial dysfunction, inflammatory infiltration, pulmonary vascular remodeling, chronic inflammation, and genetic predisposition.

PAH pathology includes medial hypertroPAHy, intimal fibrosis, plexiform lesions, venous muscularization, organized thrombi, and vascular destruction. Diagnosing PAH in COPD patients without right heart failure signs is challenging, requiring objective tests like echocardiograPAHy.[14]Hypoxemia presence should warrant further investigation.[15]

## CONCLUSION

The frequency of pulmonary hypertension is similarly prevalent among individuals exposed to tobacco smoke and biomass fuel. However, a significant increase in pulmonary hypertension frequency is observed among those exposed to biomass smoke for over 10 years. Therefore, biomass exposure should be recognized as a crucial etiological agent in the development of COPD.

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