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ORIGINAL ARTICLE

# Comparison of tobacco induced versus biomass induced chronic obstructive pulmonary disease patients during acute exacerbations.

**Saima Akhter[1], Sadhna Priya[2], Nausheen Saifullah[3], Naseem Ahmed[4]**

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**ABSTRACT… Objective:** To investigate the differences in clinical characteristics and in-hospital outcomes between BM-COPD and T-COPD during exacerbation. **Study Design:** Prospectively study. **Setting:** Jinnah Post Graduate Medical Center, Karachi. **Period:** January to December 2018. **Material & Methods:** One hundred and fifty seven consecutive patients with acute exacerbation of COPD were study. They were categorized into two groups taking into account the exposure to tobacco smoke or biomass. Clinical features and outcomes were evaluated into both groups. Data was entered in SPSS version 21. **Results:** Total 151 participants were recruited into the study with 100 (66.2%) participants in smoking group and 51(33.8%) participants biomass exposure group. Overall median age of patients was 65 (IQR=56 – 70) years. Age was not significantly different among two exposure group (p=0.506). Proportion of females were significantly higher in Biomass group (p<0.001). None of the biomarker was significantly different at the time of presentation among two groups. History of ischemic heart disease was more prevalent in biomass exposure than smoking group (p=0.016). Initial response to BiPAP was better in tobacco induced group at 24 hours because improvement in PaCo2 and heart rate was seen (p0.014) but overall mortality and morbidity was same. Among biomass exposure there were 8 (16.3%) mortalities while mortalities in smoking group were 15 (15.3%) and statistically the difference was not significant (p=0.026). **Conclusion:** Biomass-induced COPD is more prevalent in female patients, with comorbid in the form of Ischemic Heart Disease. The present study demonstrated that patients with BM-COPD and T-COPD during their acute exacerbation have similar mortality. Therefore clinicians should start the same standard treatment in any patient with BM-COPD patient during exacerbation as validated in T-COPD.

**Key words:** Acute Exacerbation of COPD, Biomass-COPD, Mortality, Morbidity, Tobacco Induced COPD.

## INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is a major healthcare concern worldwide[1] and is one of the leading causes of mortality and morbidity. It represents a huge economic and social burden.[1,2] It is characterized by persistent airflow limitation and respiratory symptoms.[2,3] Tobacco smoke is considered as main risk factor for Tobacco induced Chronic Obstructive Pulmonary Disease (T-COPD) in developed countries[2,3], however cigarette smoking is not the sole cause for COPD[4,5], Biomass smoke (BM) exposure has large contribution in causing Biomass induced Chronic Obstructive Pulmonary Disease (BM-COPD) in developing countries, particularly in women.[1,2] Biomass fuels includes use of woods, animal dung, coal and crop residues, and animal dung for cooking purposes. The main harmful components of such smoke are oxysulfide, oxynitride, oxycarbide, incompletely burned hydrocarbon particles, and multicyclic organic compounds.[2,6] Despite the huge burden of B-COPD, this respiratory condition was initially underappreciated worldwide.[1,2] But in recent years, data addressing the role of biomass fuels in the pathogenesis of COPD and its impact on patient's life have received increasing attention.[4,7,8,9]

COPD patients have frequent episodes of acute exacerbations, that may need hospitalization.[8] Acute exacerbation of Chronic Obstructive

1. FCPS, Assistant Professor Chest Medicine, Liaquat National Hospital, Karachi.
2. MBBS, Postgraduate Trainee Chest Medicine, Jinnah Postgraduate Medical Center, Karachi.
3. FCPS, Associate Professor Chest Medicine, Jinnah Postgraduate Medical Center, Karachi.
4. FCPS, Assistant Professor Chest Medicine, Jinnah Postgraduate Medical Center, Karachi.

**Correspondence Address:**
Dr. Saima Akhter
Department of Chest Medicine
Liaquat National Hospital.
drsaima_82@hotmail.com

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Pulmonary Disease (AECOPD) is defined as "an acute worsening in the patient's condition from the stable state, which is sustained and may warrant the patient to seek additional treatment". AECOPD is thoroughly investigated in last four decades and there are predefined parameters to identify the initial severity and predict the mortality and morbidity[9,10,11] but as per author's best knowledge there are no current publish data to separately address the mortality in acute exacerbation on BM-COPD. Hence, little is known about the clinical course and outcomes during exacerbation in BM-COPD or their similarities and differences compared with T-COPD. To address the issue, we undertook a prospective observational study to investigate in detail the differences in clinical features and in-hospital outcomes between T-COPD and BM-COPD during exacerbation.

## MATERIAL & METHODS

The study was conducted in JPMC, Karachi. The research team recruited consecutive patients admitted with AECOPD from December 2018 to December 2019 at the Chest Medicine department. Informed consent was taken from either the patient or nexttokin in case where patients were unable to consent because of low GCS or vital instability. AECOPD was diagnosed clinically upon admission as defined by Anthonisen criteria (a) worsening dyspnea (b) increase in purulence of sputum (c) increase in volume of sputum. Patients with concomitant diagnosis of other respiratory diseases: Interstitial Lung Disease, pneumothorax, bronchiectasis, parenchymal disease secondary to previous Tuberculosis, and patients with TYPE II respiratory failure secondary to musculoskeletal disease or neurological diseases were excluded. Patients were classified into two groups: Tobacco induce COPD (T-COPD) are the patients who have smoking background with a pack-year index above than 10 or biomass induce COPD (BM-COPD) patients who never smoke but with history of significant exposure to any kind of biomass. It was difficult to quantify the cumulative exposure of biomass smokes, because most of patients had exposure during their youth and time duration between symptoms and that exposure was more than 15 years. Secondly the stoves mostly are placed in open air in summer while during winters they relocate to close kitchens. For the purpose of this study, we considered a history of at-least 30 mins per day for more than 5 years of biomass exposure as significant.[1,9]

The study was approved by the Institutional review board of JPMC. (F.2-81/2020-GENL/42969/JPMC)

Demographic details including age, gender, occupation, smoking status, accompanying chronic diseases and comorbidities were asked. Clinical data includes pretreatment laboratory test results, including hematocrit, SGPT, Arterial Blood Gases, and SO2 at baseline, 2 to 6 hours and 24 hours of BIPAP were recorded, along with Heart rate and mean arterial pressure monitoring. Type II respiratory failure was defined as PCO2 of more than 45mmHg. These Patients were admitted in ward and ICU accordingly, and Bilevel Positive Airway Pressure (BiPAP) was applied via face mask at time of admission, with settings progressively escalating to maximum of IPAP of 24 cmH2O and EPAP of 12 cmH2O while closely observing patient comfort. Oxygenation provided to maintain SaO2 between 88 to 92%. Information concerning the in-hospital duration and mortality and improvement in clinical and lab parameters were compared in both groups. Standard treatment of AECOPD including Systemic steroids, Nebulised short-acting B 2 agonists (SABA) and steroids were provided to all patients.

Data was entered in SPSS version 21 for statistical analysis. Categorical variables were presented as frequency and percentage. Numerical variables were presented as mean ± standard deviation when normally distributed otherwise summarized as median with inter-quartile range (IQR). Assumption of normality was tested using Shaprio-wilk test. Chi-square/ Fisher-exact test was applied to compare categorical variables among two groups. Independent t-test or Man-Whitney U test was applied to compare numerical variables among the two exposure groups. Wilcoxon sign rank test was applied to compare numerical variables at baseline and at 24 hours.

P-value ≤0.05 was taken as statistically significant

## RESULTS

Patients' socio-demographic and baseline characteristics among two exposure are presented in Table-I. Total 151 participants were recruited into the study with median (IQR) age of 65 (56 – 70) years. Among 151 participants, more than half had the history of smoking exposure (n=100, 66.2%). Age was not significantly different among two exposure groups (p=0.506). Proportion of male gender was significantly higher in smoking group (n=92, 92%) than patients who were exposed to biomass (n=8, 15.7%) (p<0.001).

Table-II shows comparison of baseline clinical parameters among two exposure groups. None of the clinical parameter at baseline was significantly different among two groups. Out 151 patients, majority were discharged alive (n=124, 82.1%) and few left against medical advice (n=4, 2.6%) and 23 (15.2%) were in-hospital mortalities. Among biomass exposure there were 8 (16.3%) mortalities while mortalities in smoking group were 15 (15.3%) and the difference was not significant (p=0.872).

After 24 hours, Median $PaCO_2$ was significantly higher in biomass group than smoking group (p=0.014) (Figure-1). Median heart rate in biomass group was significantly lower than smoking group (p=0.034) (Figure-2). Initial response to BiPAP was better in tobacco induced group at 24 hours because improvement in PaCo2 and heart rate was seen (p0.014) but overall mortality and morbidity was same. Table-III depicts comparison of clinical parameters at baseline and 24 hours for smoking and biomass group. In smoking group, there was significant difference in pH, PaCO2, HCO3, SO2, and respiratory rate respiratory rate at baseline and 24 hours and these parameters were also significantly different for biomass group at baseline and 24 hours.

| Study Characteristics | Smoking N=100 | Biomass N=51 | P-Value |
|---|---|---|---|
| Age | 64.5 (56.5 - 70) | 65 (56 - 80) | 0.506 |
| Gender, male (%) | 92 (92) | 8 (15.7) | **<0.001 |
| Diabetes mellitus | 11 (11) | 6 (11.8) | 0.888 |
| Chronic kidney disease | 1 (1) | 1 (2) | 1.00 |
| Hypertension | 33 (33) | 23 (45.1) | 0.146 |
| Ischemic heart disease | 1 (1) | 4 (7.8) | *0.045 |
| NIV applied | 88 (88) | 47 (92.2) | 0.433 |
| HCT | 42.3 (37.25 - 48.47) | 42.1 (36.1 - 49.2) | 0.954 |
| Platelets | 226500 (168500 - 306000) | 257000 (178000 - 341000 | 0.075 |
| TLC | 12.80 (9.28 - 17.35) | 13 (8.1 - 16.8) | 0.865 |
| N% | 86 (79 - 92) | 84 (78 - 90) | 0.391 |
| L% | 10 (5 - 15) | 10 (6.6 - 16) | 0.942 |
| Urea | 50 (35 - 68.75) | 45 (30 - 60) | 0.122 |
| Creatinine | 0.98 (0.67 - 1.32) | 0.80 (0.63 - 1.31) | 0.152 |
| Sodium | 140 (135.25 - 143) | 139 (135 - 142) | 0.878 |
| Potassium | 4.1 (3.7 - 4.7) | 4.30 (3.90 - 4.60) | 0.273 |
| Bicarbonate | 29.95 (25 - 35.15) | 31.5 (25.2 - 36) | 0.455 |
| Chloride | 98 (93 - 103) | 99 (92 - 103) | 0.522 |
| Tot-billi | 0.51 (0.36 - 0.78) | 0.49 (0.3 - 0.88) | 0.332 |
| SGPT | 32 (20 - 54) | 28 (17 - 42) | 0.529 |
| PT | 11.8 (11 - 13.97) | 11.30 (10.80 - 12.30) | 0.33 |
| INR | 1.10 (0.97 - 1.33) | 1.07 (0.91 - 1.30) | 0.533 |

**Table-I. Comparison of socio-demographic and clinical presentation of patients among exposure groups**
Note: All quantitative variables are expressed as median (inter-quartile range)
*Significant at p<0.05, **Significant at p<0.01

| | Smoking | Biomass | P-Value |
|---|---|---|---|
| pH | 7.28 (7.23 - 7.34) | 7.28 (7.24 - 7.34) | 0.865 |
| PaCO2 | 65.3 (54.25 - 82) | 66.30 (55.30 - 80) | 0.954 |
| PaO2 | 60.95 (45.25 - 85.75) | 59.1 (45 - 71) | 0.607 |
| HCO3 | 30.70 (26.05 - 36.15) | 31 (25.70 - 35.60) | 0.957 |
| SO2 | 87 (70.77 - 92.45) | 88 (72 - 90) | 0.452 |
| Heart rate | 88 (97.5 - 101) | 90 (84 - 105) | 0.954 |
| Respiratory rate | 28 (24 - 31.5) | 28 (25 - 32) | 0.971 |

**Table-II. Comparison of baseline clinical parameters among two exposure groups**

Note: All quantitative variables are expressed as median (inter-quartile range)
*Significant at p<0.05,

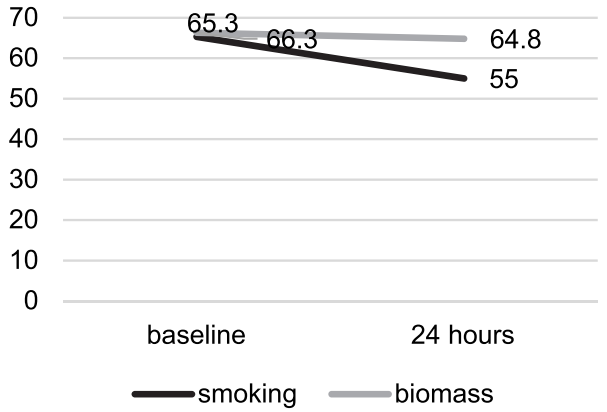


**Figure-1. Comparison of PaCO2 between smoking and biomass at baseline and 24 hours**

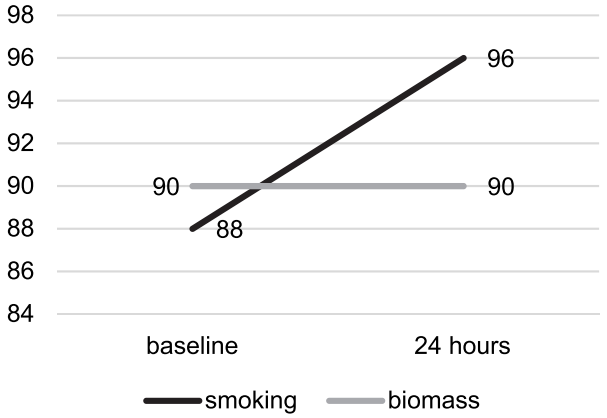


**Figure-2. Comparison of PaCO2 between smoking and biomass at baseline and 24 hours**

| | Smoking | | | Biomass | | |
|---|---|---|---|---|---|---|
| | Median (IQR) | Range (Min - Max) | P-Value | Median (IQR) | Range (Min - Max) | P-Value |
| Baseline pH | 7.28 (7.23 - 7.34) | 7 - 7.54 | **<0.001 | 7.28 (7.24 - 7.34) | 6.79 - 7.49 | **<0.001 |
| 24 hours pH | 7.38 (7.33 - 7.43) | 6.85 - 7.56 | | 7.37 (7.33 - 7.41) | 7.12 - 7.65 | |
| baseline PaCO2 | 65.3 (54.25 - 82) | 16.30 - 165 | **<0.001 | 66.30 (55.30 - 80) | 26 - 119 | **0.007 |
| 24 hours PaCO2 | 55 (47.77 - 65.07) | 27 - 141 | | 64.8 (54 - 71.3) | 30.60 - 100 | |
| baseline PaO2 | 60.95 (45.25 - 85.75) | 20.80 - 213 | 0.128 | 59.1 (45 - 71) | 17 - 197 | 0.094 |
| 24 hours PaO2 | 71.5 (59 - 86) | 31 - 117 | | 73.1 (57 - 88) | 30 - 224 | |
| baseline HCO3 | 30.70 (26.05 - 36.15) | 16 - 99.50 | **<0.001 | 31 (25.70 - 35.60) | 12.50 - 46.50 | **<0.001 |
| 24 hours HCO3 | 34 (32 38.2) | 19.50 - 88 | | 34 (28.02 - 38.57) | 17.50 - 49.50 | |
| baseline SO2 | 87 (70.77 - 92.45) | 24.80 - 99 | **<0.001 | 88 (72 - 90) | 34.20 - 99.30 | **0.001 |
| 24 hours SO2 | 91 (86 - 96.9) | 50 - 99.80 | | 90.5 (86.25 - 94) | 56 - 99 | |
| baseline heart rate | 88 (97.5 - 101) | 56 - 126 | 0.177 | 90 (84 - 105) | 62 - 160 | 0.270 |
| 24 hours heart rate | 96 (88 - 100) | 58 - 128 | | 90 (86 - 98) | 68 - 120 | |
| baseline respiratory rate | 28 (24 - 31.5) | 14 - 143 | **<0.001 | 28 (25 - 32) | 21 - 40 | **<0.001 |
| 24 hours respiratory rate | 24 (22 - 28) | 16 - 38 | | 24 (24 - 26) | 20 – 38 | |

**Table-III. Comparison of patients' clinical parameters between baseline and 24 hours**

## DISCUSSION

The present study investigated the differences in clinical features and outcomes between T-COPD and BM-COPD during exacerbation. AECOPD has very important impact in overall management and outcomes of patients with COPD.[7] These episodes have negative impact on overall health status of patient by affecting the disease progression and mortality.[10,11] It is therefore important to identify in-hospital mortality rate to develop best management strategy of these patients. We aimed to determine that if the current practices of AECOPD management are equally useful in BS-COPD and the prognosis and in hospital course are comparable in both groups.

There are few studies that have compared the overall survival among stable patients from two groups[12] however no studies have investigated the in-hospital mortality and compare the outcomes of these two groups during exacerbation. Though data from Mexican cohort represents similarity of clinical characteristics and long-term mortality between BS-COPD and T-COPD, but data was from stable patients.[12] To authors' best knowledge, ours is the first prospective study that compares the clinical, laboratory features and in-hospital between two groups during exacerbation.

Patients in BM-COPD group were predominantly women, and female predominance in this group is well documented in literature.[13] Literature proved that there are Several Comorbidities, associated with COPD and impact over outcome as well as the patients' quality of life.[14] It is interesting to note that the comorbidities among the two groups was not significantly different in present study except for the history of ischemic heart disease was higher in biomass exposure group that is contradicting the previous studies.[15] A previous report demonstrated higher levels of IL-8 and IL-6 in T-COPD when compared to BM-COPD and suggested a role of these markers in development of atherosclerosis.[15,16] Solleiro-Villavicencio and colleagues has also found a higher prevalence of ischemic heart disease in female patients with T-COPD when compared to BM-COPD.[14] The possible explanation to this discrepancy between present study and previous studies can be the small sample size, and use of different sources of biomass fuels.[17] Animal models demonstrated that when mice were exposed to biomass fuels for long time, their inflammatory response might differ with different kinds of fuels. used so the systemic involvement.[17]

Present study demonstrated no significant difference in baseline (on admission) and after 24 hours of admission vital monitoring and ABGs. Median $PaCO_2$ in biomass group which was higher than smoking group that shows the late response of BM-COPD group when compared to T-COPD group but this doesn't affect in-hospital course of overall mortality of patients. Our finding is consistent with previous studies reporting no difference in overall mortality of patients.[12] On combining our findings with that previous cohort exposed to two different kinds of smokes, we can conclude that difference in pathology[18], their nature of exposure and phenotype may not lead to difference in their management and overall outcomes.

This current study has few limitations. The most obvious is that it's a single centered study but JPMC is one of the largest pulmonary centers of country and it caters a diverse population from different backgrounds and therefore represented a large spectrum of population. The possibility of recall bias in BS-COPD can't be ignored as the data was asked from the patient after considerable time lapse. More prospective studies from other part of World will be needed to validate the results. The strength of study is that evaluation and treatment of all the patients was done by same team. There was no difference in the treatment pattern of any patient.

Currently there is no difference in AECOPD treatment between T-COPD and BM-COPD. It still remain to seen if there is difference in long term mortality and number of exacerbation between two groups.

## CONCLUSION

Biomass-induced COPD is more prevalent in female patients, with comorbid in the form of Ischemic Heart Disease. The present study

demonstrated that patients with BM-COPD and T-COPD during their acute exacerbation have similar mortality. Therefore clinicians should start the same standard treatment in any patient with BM-COPD patient during exacerbation as validated in T-COPD.



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## AUTHORSHIP AND CONTRIBUTION DECLARATION

| No. | Author(s) Full Name | Contribution to the paper | Author(s) Signature |
|---|---|---|---|
| 1 | Saima Akhter | Study design, Draft writing. | |
| 2 | Sadhna Priya | Data collection, Draft writing. | |
| 3 | Nausheen Saifullah | Study design, Proof reading. | |
| 4 | Naseem Ahmed | Data collection, Proof reading. | |