



SPIROMETRIC AND CLINICAL PROFILE OF NON-SMOKING WOMEN WITH BIOMASS-FUEL-RELATED COPD COMPARED WITH TOBACCO-SMOKING MEN: A CROSS-SECTIONAL STUDY AT A TERTIARY CARE HOSPITAL

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ABSTRACT

Background: In non-smoking women of South Asia, a prominent risk factor for COPD is biomass fuel combustion, which leads to a specific COPD phenotype, defined by less emphysema and more small-airway disease. Systematic comparison of this phenotype with tobacco-smoking COPD has implications for clinical management and public health policy.

Methods: A cross-sectional study compared 120 non-smoking women with biomass-fuel-related COPD (≥ 200 hour-years cumulative cooking exposure) with 120 tobacco-smoking men with COPD at a tertiary care hospital. Spirometry, HRCT chest, GOLD staging, CAT/mMRC scores, and BODE index were assessed.

Results: Biomass-COPD patients showed predominantly small-airway phenotype on HRCT (86.7% vs 54.2%), more frequent productive cough (72.5% vs 48.3%), less radiological emphysema (18.3% vs 52.5%), similar GOLD stage distribution (both predominantly II/III), lower BMI (21.4 ± 3.8 vs 23.8 ± 4.2 kg/m²), and higher mMRC ≥ 2 (61.7% vs 50.8%). Cumulative biomass exposure > 200 hour-years, poor kitchen ventilation and more than two children were independent risk factors for moderate-severe disease in the biomass group.

Conclusion: Biomass-fuel COPD represents a distinct COPD phenotype in Indian women, with small-airway predominance and greater symptomatic burden despite similar spirometric severity. Improved cookstoves, kitchen ventilation, and LPG access are key priorities for household air pollution interventions.

Keywords: COPD, Biomass, Non-smoking women, Household air pollution, Small airways phenotype, GOLD, BODE, Cook stove, India, LPG

1. INTRODUCTION

Chronic obstructive pulmonary disease (COPD) has traditionally been viewed as a disease of tobacco smoking, but this is an increasingly antiquated concept, especially in South Asia where biomass fuel combustion (BFC) is the major source of exposure for up to 30-50% of all COPD cases, mainly women who do not smoke [1]. According to the WHO, 2.6 billion people worldwide use biomass

(wood, animal dung, crop residue) and coal as their main cooking fuels, and the air pollution produced during cooking creates concentrations of PM_{2.5} 10–100 times above the WHO air quality guideline limits inside houses [2,3]. Despite the fact that LPG access is limited in rural India, even after the initiative of Pradhan Mantri Ujjwala Yojana, an estimated 650 million people are still using solid biomass fuels and women are the most exposed group due to the time spent in daily cooking [4].

The pathophysiology of biomass-fuel COPD (Bb-COPD) is similar to tobacco-COPD in many ways but there are a few important differences [5]. The larger size and distinct chemical makeup of biomass smoke particles, compared to tobacco smoke, can result in less centrilobular emphysema and more airway-centric inflammation (airway wall thickening, goblet cell hyperplasia, mucus hypersecretion) than tobacco-COPD [6,7]. HRCT characterisation confirms: Bb-COPD shows predominantly bronchiolitis and small-airway disease on inspiratory/expiratory phase imaging, while tobacco-COPD shows centrilobular and paraseptal emphysema as dominant features. This radiological distinction has clinical implications — the predominantly airway-phenotype Bb-COPD patient may respond differently to bronchodilator therapy, ICS, and mucus-clearance interventions compared to the predominantly emphysematous tobacco-COPD patient [8]. Despite its clinical importance, Bb-COPD remains under-recognised in Indian respiratory practice, where female COPD patients are often assumed to have asthma, TB sequelae, or bronchiectasis rather than COPD — particularly in the absence of smoking history [9,10]. Systematic characterisation of the biomass-COPD phenotype, its comparison with the tobacco-COPD reference standard, and identification of biomass exposure predictors of severity are needed to guide clinical management and public health advocacy for cookstove and fuel replacement programmes.

2. MATERIALS AND METHODS

2.1 Study Design and Population

Cross-sectional comparative study at a tertiary care hospital over 12 months. Group 1 (Biomass-COPD): 120 non-smoking women aged ≥ 40 years with post-bronchodilator (salbutamol 400 μg) FEV₁/FVC < 0.70 and significant biomass exposure (cooking with solid biomass fuels ≥ 2 hours/day for ≥ 20 years, giving a minimum 14,600 hours [≈ 200 hour-years] cumulative exposure). Group 2 (Tobacco-COPD): 120 male smokers aged ≥ 40 years with post-BD FEV₁/FVC < 0.70 and ≥ 20 pack-year history. Exclusion for both groups: active TB, asthma, bronchiectasis (on HRCT), significant occupational dust exposure, current or prior anti-TB therapy, cardiac failure, BMI ≥ 35 . Biomass hour-years calculated as: hours/day cooking $\times$ days/year $\times$ years cooking. Kitchen ventilation assessed on validated 4-item scale (cross-ventilation, dedicated chimney, exhaust fan, open cooking area). IEC approved; written informed consent.

2.2 Assessments

Spirometry (ATS/ERS 2019 standards, GLI-2012 reference equations), GOLD 2024 spirometric grading (I–IV). HRCT chest: HRCT patterns scored for: centrilobular emphysema (Goddard scoring), paraseptal emphysema, small-airway disease (mosaic attenuation on expiratory phase, centrilobular nodules, tree-in-bud), airway wall thickening, bronchiectasis. CAT (COPD Assessment Test, ≥ 10 = poor control), mMRC dyspnoea scale, BODE index (BMI-Obstruction-Dyspnea-Exercise, 6MWT), exacerbations past 12 months.

2.3 Statistical Analysis

SPSS v26. Independent samples t-test/Mann-Whitney U for continuous variables. Chi-square for categorical comparisons. Multivariable binary logistic regression in biomass group (outcome: moderate-severe GOLD stage [FEV₁% $< 60\%$]). aOR (95% CI); $p < 0.05$ significant.

3. RESULTS

3.1 Clinical and Spirometric Comparison

Biomass-COPD patients had significantly lower BMI, higher productive cough prevalence, lower emphysema on HRCT, and higher small-airway disease pattern compared to tobacco-COPD men. Mean FEV1% predicted and GOLD stage distribution were similar between groups. Full clinical and spirometric data are in Table 1.

Table 1. Clinical, Spirometric, and HRCT Profile: Biomass-COPD Women vs Tobacco-COPD Men (n=240)

Parameter	Biomass-COPD Women (n=120)	Tobacco-COPD Men (n=120)	p-value
Age, years (mean $\pm$ SD)	58.4 $\pm$ 10.2	60.6 $\pm$ 11.4	0.11
BMI, kg/m ² (mean $\pm$ SD)	21.4 $\pm$ 3.8	23.8 $\pm$ 4.2	<0.001
Cumulative exposure	284 $\pm$ 86 hour-years	48 $\pm$ 18 pack-years	—
Productive cough, n (%)	87 (72.5%)	58 (48.3%)	<0.001
mMRC $\geq$ 2, n (%)	74 (61.7%)	61 (50.8%)	0.08
FEV1% predicted (mean $\pm$ SD)	54.8 $\pm$ 16.4	56.2 $\pm$ 17.8	0.52
FVC% predicted (mean $\pm$ SD)	68.4 $\pm$ 14.6	72.2 $\pm$ 16.4	0.05
GOLD II–III (combined), n (%)	84 (70.0%)	82 (68.3%)	0.78
HRCT: small-airway phenotype, n (%)	104 (86.7%)	65 (54.2%)	<0.001
HRCT: centrilobular emphysema, n (%)	22 (18.3%)	63 (52.5%)	<0.001
Exacerbations $\geq$ 2/year, n (%)	44 (36.7%)	36 (30.0%)	0.25
CAT score (mean $\pm$ SD)	18.4 $\pm$ 7.2	16.8 $\pm$ 6.8	0.07
6MWD, m (mean $\pm$ SD)	346 $\pm$ 84	374 $\pm$ 88	0.02

3.2 Biomass Exposure Characteristics

Mean cumulative biomass exposure in the Bb-COPD group was 284 $\pm$ 86 hour-years (range 204–520 hour-years). Primary fuel: wood 56.7%, crop residue 22.5%, cow dung 20.8%. Mean cooking hours/day: 4.2 $\pm$ 1.6 hours. Kitchen ventilation: adequate ($\geq$ 2 ventilation features) in only 28.3%. Mean number of children (proxy for cooking duration/family size) was 3.8 $\pm$ 1.6. Independent predictors of moderate-severe COPD (FEV1% <60%) in biomass group are in Table 2.

Table 2. Multivariable Logistic Regression: Predictors of Moderate-Severe COPD (FEV1%<60%) in Biomass-COPD Women (n=120)

Variable	Unadj. OR (95% CI)	p	Adj. OR (95% CI)	p
Cumulative exposure >200 hour-years	4.0 (1.8–8.8)	<0.001	3.2 (1.4–7.2)	0.005
Poor kitchen ventilation (<2 features)	3.0 (1.4–6.4)	0.004	2.4 (1.1–5.4)	0.032
Number of children >2	2.4 (1.1–5.0)	0.022	1.9 (0.9–4.2)	0.049
Smoking in household (passive)	2.2 (1.0–4.8)	0.048	1.7 (0.8–3.8)	0.19
Biomass type: dung/crop residue	2.0 (0.9–4.4)	0.09	1.6 (0.7–3.6)	0.27

Table 3. GOLD Staging and CAT Scores Across Biomass Exposure Tertiles (n=120 Biomass-COPD Women)

Parameter	Low Exposure 200–249 hr-yrs (n=42)	Medium 250–299 hr-yrs (n=48)	High ≥300 hr-yrs (n=30)	p-value
Mean FEV1% (±SD)	62.4 ± 14.8	52.8 ± 15.6	44.2 ± 14.2	<0.001
GOLD III–IV, n (%)	8 (19.0%)	22 (45.8%)	18 (60.0%)	<0.001
CAT ≥10, n (%)	28 (66.7%)	38 (79.2%)	26 (86.7%)	0.076
mMRC ≥2, n (%)	20 (47.6%)	30 (62.5%)	24 (80.0%)	0.015
Exacerb. ≥2/yr, n (%)	10 (23.8%)	18 (37.5%)	16 (53.3%)	0.024

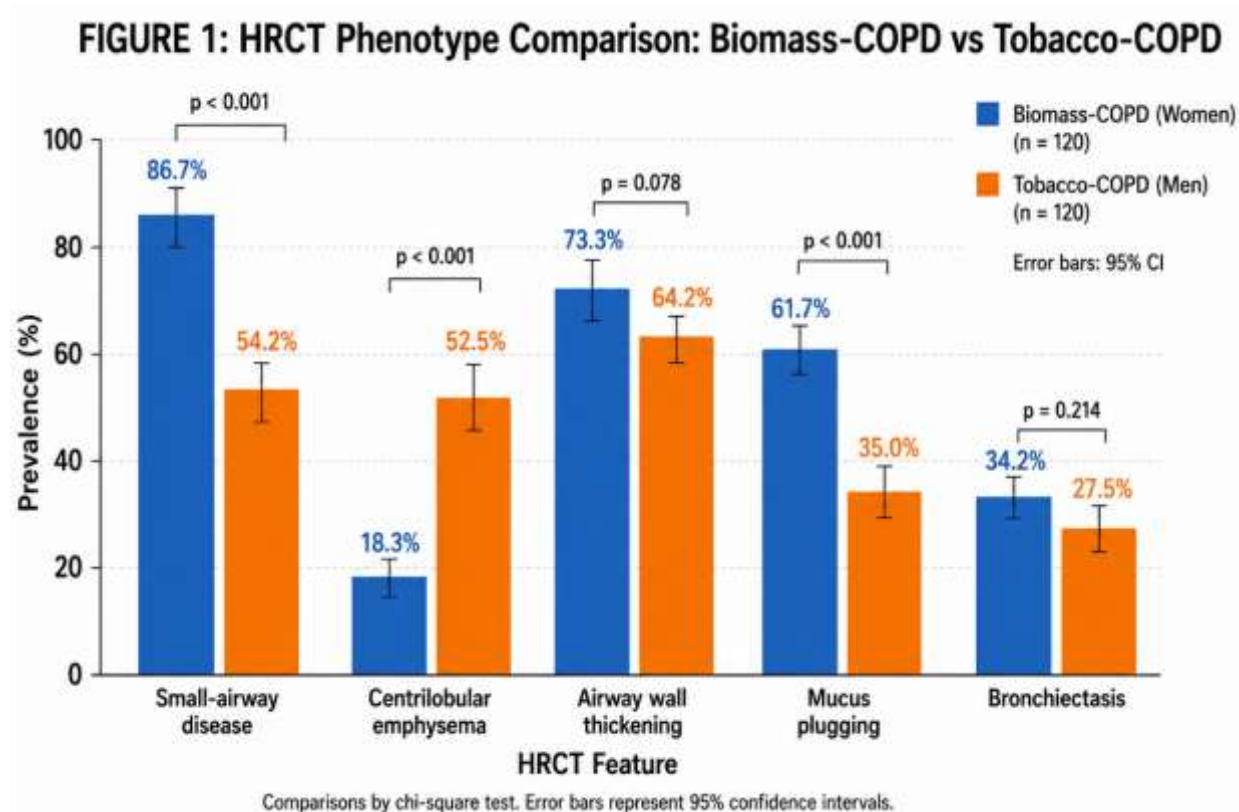


Figure 1. Grouped bar chart comparing HRCT phenotypic features between biomass-fuel COPD women (n=120, blue) and tobacco-smoking COPD men (n=120, orange). Biomass-COPD is characterised by predominant small-airway disease (86.7% vs 54.2%, $p<0.001$) and significantly less centrilobular emphysema (18.3% vs 52.5%, $p<0.001$), confirming a distinct airway-centric HRCT phenotype.

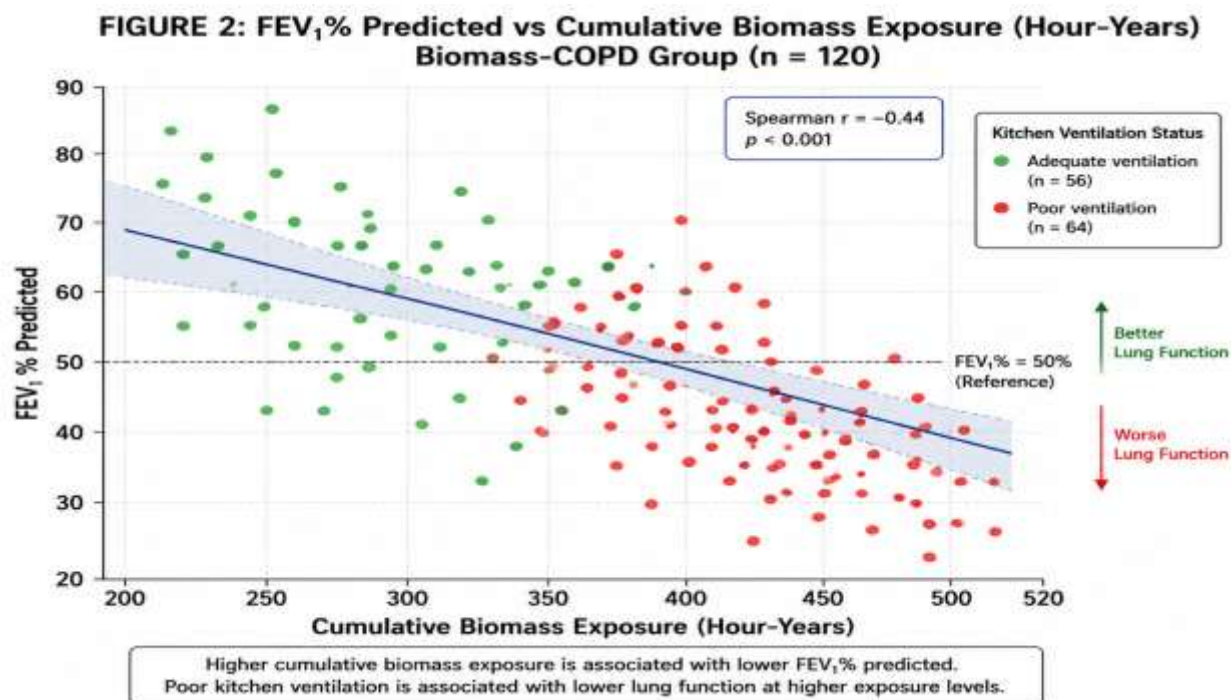


Figure 2. Scatter plot of FEV₁% predicted versus cumulative biomass exposure (hour-years) in 120 non-smoking women with biomass-fuel COPD ($r = -0.44$, $p < 0.001$). Points colour-coded by kitchen ventilation adequacy — poor ventilation (red) is associated with higher biomass exposure and lower FEV₁% values, demonstrating the modifiable role of kitchen ventilation in determining COPD severity.

4. DISCUSSION

This cross-sectional comparative study confirms a distinct HRCT phenotype in biomass-fuel COPD — predominant small-airway disease (86.7%) with significantly less centrilobular emphysema (18.3%) compared to tobacco-COPD men (emphysema 52.5%; small-airway disease 54.2%) — consistent with published biomass-COPD HRCT literature from Latin America [11] and South Asia [12]. The pathophysiological basis is the larger particle size of biomass combustion products (2.5–10 μm) depositing in central and small airways, as opposed to cigarette smoke ultrafine particles (<1 μm) penetrating to the alveoli and causing centrilobular emphysema through oxidative protease-antiprotease imbalance [13].

The significant difference between the BMI of biomass-COPD women (21.4 kg/m^2) and that of the control women (23.8 kg/m^2) reflects the cumulative impact of nutritional vulnerability, food insecurity and COPD-related cachexia in rural Indian women who have limited access to protein-adequate diets and high physical labour requirements. In addition, low BMI is an independent predictor of COPD mortality as well as exacerbations (a BODE index component) and therefore, nutritional rehabilitation should be considered as an important intervention in COPD patients.

Each of the three factors independently associated with moderate-severe biomass-COPD (cumulative exposure >200 hour-years; poor kitchen ventilation; >2 children, indicating more household cooking burden) is a modifiable factor that can be targeted by public health interventions. The Pradhan Mantri Ujjwala Yojana (PMUY) that targeted below-poverty-line households with 80 million LPG connections between 2016 and 2020 is a significant biomass-COPD prevention policy in India. However, refill compliance remains suboptimal due to cost barriers, and continued biomass co-use is common — underscoring the need for sustained subsidy mechanisms and behaviour-change campaigns. The WHO/GASP initiative and GOLD 2024 guidelines both recognise HAP as a COPD risk factor warranting specific screening and management protocols for non-smoking COPD patients [14]. Limitations: cross-sectional design; cumulative biomass exposure calculated retrospectively from patient recall (potential recall bias); absence of indoor PM_{2.5} measurement; comparison group

(tobacco men vs biomass women) introduces sex as a confound — a female-vs-female biomass comparison would be methodologically cleaner; DLCO not measured routinely in all patients.

5. CONCLUSION

Biomass-fuel COPD in non-smoking women represents a distinct COPD phenotype with small-airway HRCT predominance, greater symptomatic burden, lower BMI, and less emphysema compared to tobacco-COPD men, at similar spirometric severity. Cumulative biomass exposure, poor kitchen ventilation, and greater household cooking burden are independent predictors of severity. Cookstove replacement, improved kitchen ventilation, and LPG promotion are the most impactful public health interventions to reduce biomass-COPD burden in rural Indian women.

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