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Original Research Article

Clinical Severity, Exacerbation Frequency and Radiological Findings: A Comparative Study of Smoking-Related versus Biomass-Related Chronic Obstructive Pulmonary Disease

Ravishek Kumar Verma[1], Abhijeet Kumar[2], Saksham Prateek[3]

[1]Academic Junior Resident (2nd Year), Department of Respiratory Medicine, Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India

[2]Academic Junior Resident (1st Year), Department of Respiratory Medicine, Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India

[3]Junior Resident (1st Year), Department of Respiratory Medicine, Jawaharlal Nehru Medical College & Hospital, Bhagalpur, Bihar, India

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Corresponding Author: Dr. Ravishek Kumar Verma

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Abstract

Background: While tobacco smoking has long been the dominant risk factor discussed in COPD literature, biomass fuel combustion has steadily emerged as an equally important — and arguably underappreciated — aetiological driver, particularly in rural South Asian populations. The two exposure types appear to produce phenotypically different disease, yet comparative data from resource-limited settings in eastern India remain sparse.

Aim: We aimed to compare clinical severity, exacerbation burden, spirometric parameters, and radiological characteristics between smoking-related COPD (S-COPD) and biomass-related COPD (B-COPD) in a tertiary care setting in Bihar.

Methods: A cross-sectional comparative study was conducted at the Department of Respiratory Medicine, JLNMCH, Bhagalpur, between August 2025 and July 2026. A total of 110 COPD patients diagnosed per GOLD 2025 criteria (post-bronchodilator $FEV_1/FVC < 0.70$) were enrolled — 55 with exclusive tobacco exposure (S-COPD) and 55 with exclusive biomass fuel exposure (B-COPD). Baseline demographics, spirometry, GOLD grading, CAT score, mMRC dyspnoea grade, annual exacerbation count, hospitalisation rates, and radiological findings on chest X-ray and HRCT were recorded and compared between the groups.

Results: S-COPD patients were mostly male (92.7%) and somewhat older (mean 62.4 ± 8.7 years), whereas the B-COPD group was predominantly female (74.5%) and from rural areas (89.1%). Spirometrically, S-COPD showed lower post-bronchodilator FEV_1 (46.8 ± 14.2% vs 51.6 ± 13.7%; p=0.049), higher CAT scores (22.7 ± 5.4 vs 20.1 ± 5.8; p=0.019), more frequent exacerbations (mean 2.6 ± 1.2 vs 1.8 ± 1.0; p=0.001), and a higher hospitalisation rate over the preceding year (56.4% vs 32.7%; p=0.011). More S-COPD patients fell in GOLD Group E (50.9% vs 23.6%; p=0.003). HRCT findings told a clear story: centrilobular emphysema dominated in S-COPD (72.7% vs 27.3%; p<0.001), while B-COPD showed significantly more bronchial wall thickening (69.1% vs 38.2%; p=0.001), air trapping (63.6% vs 45.5%; p=0.041), bronchiectasis (30.9% vs 12.7%; p=0.021), peribronchial thickening (41.8% vs 16.4%; p=0.003), and pulmonary hypertension (47.3% vs 25.5%; p=0.015).

Conclusion: Smoking-related and biomass-related COPD differ in meaningful ways — not just epidemiologically but in their clinical behaviour and imaging patterns. S-COPD tends toward more severe obstruction and more frequent exacerbations; B-COPD, despite appearing milder on spirometry, carries a higher burden of pulmonary vascular disease. Recognising these distinctions has real implications for how we approach management and prevention, especially in regions like rural Bihar where biomass dependence remains high.

Keywords: COPD; biomass fuel; tobacco smoking; GOLD severity; exacerbations; HRCT chest; Bihar; India.

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Introduction

Chronic obstructive pulmonary disease (COPD) is one of the most pressing contributors to global morbidity, currently ranked as the third leading cause of death worldwide [1]. The disease is defined by progressive airflow limitation that is largely irreversible, and it presents clinically as worsening breathlessness, chronic cough, and episodes of acute exacerbation — all of which substantially impair a

patient's quality of life and carry significant economic consequences for families and health systems alike [2]. For decades, cigarette smoking was viewed as the near-exclusive cause. That picture has now changed considerably. Biomass fuel combustion — burning wood, dung, crop waste, and charcoal for cooking and heating — is now well established as an equivalent risk factor, and in large parts of the low- and middle-income world, it may actually be the more common one [3].

The scale of biomass exposure globally is hard to overstate. Roughly 2.4 billion people rely on such fuels as their primary domestic energy source, with the heaviest concentrations in South Asia, sub-Saharan Africa, and parts of Latin America [4]. In India specifically, over 70% of rural households still use solid fuels — meaning that millions of women, who typically shoulder the cooking burden, spend hours each day breathing in a complex mixture of fine particulate matter, carbon monoxide, and polycyclic aromatic hydrocarbons in poorly ventilated kitchens [5,6]. The epidemiological consequences are visible in the data: pooled estimates put COPD prevalence at around 8% among biomass users in India versus about 1% in those using cleaner fuels [7]. Perhaps most tellingly, India's male-to-female COPD ratio sits around 1.5:1, even though the tobacco use ratio exceeds 10:1 — a discrepancy that only makes sense if biomass smoke is driving substantial disease in women who have never smoked [8].

There is growing recognition that biomass-smoke COPD (B-COPD) and tobacco-smoke COPD (S-COPD) are not simply the same disease with different exposures — they appear to be phenotypically distinct. Radiologically, B-COPD tends to produce an airway-predominant picture: bronchial wall thickening, air trapping, peribronchial fibrosis, and bronchiectasis.

Tobacco-related COPD, by contrast, characteristically destroys lung parenchyma, producing centrilobular or panlobular emphysema [9,10]. These differences have a pathological basis too — biomass smoke appears to drive lymphocyte-predominant airway inflammation and vascular remodelling, while tobacco smoke leads more toward neutrophilic alveolar destruction [11]. Clinically, patients with B-COPD may report breathlessness that seems out of proportion to their spirometric results, and they carry a disproportionately high risk of pulmonary hypertension and right heart strain [12,13].

Despite the clinical importance of these differences, studies systematically comparing the two COPD phenotypes from tertiary centres in eastern India are limited. This is a region with high biomass dependence and appreciable COPD burden from both exposures, yet it is underrepresented in the comparative literature. The GOLD 2025 report supports phenotype-guided management in COPD, but few Indian studies have rigorously applied standardised methods to compare spirometry, symptom burden, exacerbation rates, hospital admissions, and imaging findings side by side [14].

This study was set up to address that gap. Working from a single tertiary respiratory medicine unit in Bhagalpur, Bihar, we recruited patients with exclusive tobacco or exclusive biomass exposure and systematically compared their clinical, spirometric, and radiological profiles. The intent was not merely descriptive — we hoped the findings would have practical implications for how clinicians approach these two groups differently, and how policymakers think about biomass fuel as a public health issue.

Materials and Methods

Study Design and Setting: This was a hospital-based, cross-sectional, comparative observational study carried out at the Department of Respiratory Medicine, Jawaharlal Nehru Medical College & Hospital (JLNMCH), Bhagalpur, Bihar, from 1st August 2025 to 31st July 2026.

The study received prior ethical clearance from the Institutional Ethics Committee of JLNMCH, and was conducted in accordance with the Declaration of Helsinki (2013 revision). Written informed consent was obtained from all participants before enrolment.

Study Population: All patients aged ≥40 years presenting to outpatient or inpatient respiratory services with a confirmed COPD diagnosis were screened. COPD was diagnosed per GOLD 2025 criteria — persistent respiratory symptoms with relevant exposure history, confirmed by post-bronchodilator $FEV_1/FVC < 0.70$ [14].

Patients were placed into one of two groups based on their primary exposure: Smoking-related COPD (S-COPD) for those with ≥10 pack-years of tobacco use and no biomass exposure; and Biomass-related COPD (B-COPD) for lifelong non-smokers with ≥10 years of exclusive biomass fuel use for cooking, spending at least 4 hours per day in a smoke-exposed kitchen or enclosed space. Equal numbers were enrolled in each arm (n=55 per group; total n=110).

Exclusion Criteria: We excluded patients with mixed tobacco and biomass exposure, active pulmonary tuberculosis, primary bronchiectasis, interstitial lung disease, asthma without COPD, lung malignancy, and those with comorbidities that would make spirometry unsafe (e.g., recent myocardial infarction within three months). Patients who declined consent were also excluded.

Data Collection: A structured, pre-validated proforma captured sociodemographic details (age, sex, residence, socioeconomic status), exposure

history, symptom duration, and clinical findings. The mMRC scale was used to grade dyspnoea, and the COPD Assessment Test (CAT) was administered to quantify symptom burden. GOLD ABE classification was applied using spirometric grade and exacerbation history [14]. Exacerbation frequency over the preceding 12 months was documented from patient recall, corroborated against outpatient records and prescriptions. Hospital admissions over the same period were similarly recorded.

Spirometry: Post-bronchodilator spirometry was performed using a calibrated Vitalograph® Alpha spirometer (Ireland), following ATS/ERS 2019 standardisation criteria. At least three technically acceptable and reproducible manoeuvres were obtained. FEV_1 and FVC were expressed as percentages of predicted using Indian reference equations. GOLD spirometric severity grades (I–IV) were assigned based on post-bronchodilator FEV_1% predicted.

Radiological Assessment: Posteroanterior chest radiographs were obtained in all patients and reviewed for hyperinflation, bronchovascular markings, hemidiaphragm position, retrosternal space, and heart size. HRCT of the chest was performed where clinically indicated or feasible (Siemens Somatom® 16-slice CT; window level −600 HU, width 1500 HU). CT images were assessed for emphysema subtypes, air trapping, bronchial and peribronchial wall changes, bronchiectasis, mosaic attenuation, fibrosis, and pulmonary arterial calibre. A senior radiologist, blinded to exposure group, assigned the predominant CT phenotype (emphysema-predominant, airway disease-predominant, or mixed).

Statistical Analysis: Data were entered in Microsoft Excel® and analysed with IBM SPSS version 26.0. Continuous data are presented as mean ± SD and were compared using the independent t-test (or Mann–Whitney U where normality was not met on Shapiro–Wilk testing). Categorical data are presented as frequencies and percentages, compared with Chi-square or Fisher's exact test as appropriate. A two-tailed $p<0.05$ was taken as statistically significant.

Results

Baseline Demographic and Clinical Characteristics (Table 1)

One hundred and ten patients were enrolled — 55 in each group. The two groups differed substantially in baseline demographics. S-COPD patients were older (mean 62.4 ± 8.7 vs 59.1 ± 9.3 years; p=0.048) and almost exclusively male (92.7%), while B-COPD patients were predominantly female (74.5%) and rural (89.1% vs 50.9%; p<0.001) — a profile that mirrors the known epidemiology of domestic biomass fuel use in Bihar.

Cumulative tobacco exposure in S-COPD averaged 28.6 ± 9.2 pack-years, while biomass exposure duration in B-COPD was 22.4 ± 8.1 years. Cor pulmonale on echocardiography was more prevalent in B-COPD (41.8% vs 20.0%; p=0.011), as was pulmonary hypertension (47.3% vs 25.5%; p=0.015). Cough, sputum production, wheeze, hypertension, and diabetes did not differ significantly between groups.

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants

Variable	S-COPD (n=55)	B-COPD (n=55)	p-value
Age (years), mean ± SD	62.4 ± 8.7	59.1 ± 9.3	0.048*
Male, n (%)	51 (92.7%)	14 (25.5%)	<0.001*
Female, n (%)	4 (7.3%)	41 (74.5%)	<0.001*
Rural, n (%)	28 (50.9%)	49 (89.1%)	<0.001*
Urban, n (%)	27 (49.1%)	6 (10.9%)	<0.001*
BMI (kg/m²), mean ± SD	21.3 ± 3.1	22.7 ± 3.4	0.024*
Cumulative exposure, mean ± SD	28.6 ± 9.2 pack-years	22.4 ± 8.1 years	0.001*
Symptom duration (years), mean ± SD	7.4 ± 4.2	6.9 ± 3.8	0.503
Dyspnoea (mMRC ≥ 2), n (%)	39 (70.9%)	44 (80.0%)	0.253
Chronic cough, n (%)	48 (87.3%)	43 (78.2%)	0.204
Sputum production, n (%)	45 (81.8%)	36 (65.5%)	0.057
Wheeze, n (%)	32 (58.2%)	41 (74.5%)	0.072
Cor pulmonale (ECHO), n (%)	11 (20.0%)	23 (41.8%)	0.011*
Hypertension	18 (32.7%)	14 (25.5%)	0.408
Type 2 Diabetes Mellitus	12 (21.8%)	9 (16.4%)	0.475
Pulmonary hypertension	14 (25.5%)	26 (47.3%)	0.015*

SD = Standard Deviation; BMI = Body Mass Index; mMRC = Modified Medical Research Council; ECHO = Echocardiography. *p<0.05. Chi-square for categorical; independent t-test for continuous variables.

Spirometric Parameters, GOLD Classification and Exacerbation Frequency: On spirometry, S-COPD patients showed lower post-bronchodilator FEV_1% predicted (46.8 ± 14.2% vs 51.6 ± 13.7%; p=0.049) and FVC% predicted (61.3 ± 13.1% vs 66.8 ± 12.4%; p=0.027), pointing to more pronounced airflow obstruction overall. The FEV_1/FVC ratio was slightly lower in S-COPD (0.57 ± 0.09 vs 0.60 ± 0.08) though this fell short of significance (p=0.063). When GOLD grades were compared, S-COPD had a heavier concentration at Grades III and IV (65.4% vs 47.3%; p=0.038). CAT scores confirmed greater symptom impact in S-COPD (22.7 ± 5.4 vs 20.1 ± 5.8; p=0.019). The exacerbation data were particularly striking. S-COPD patients averaged 2.6 ± 1.2 exacerbations per year versus 1.8 ± 1.0 in B-COPD (p=0.001).

Over four in ten S-COPD patients (41.8%) had three or more exacerbations in the preceding year, compared with fewer than two in ten in B-COPD (18.2%; p=0.009). More than half of S-COPD patients had been hospitalised in the past 12 months (56.4% vs 32.7%; p=0.011). Unsurprisingly, GOLD Group E predominated in S-COPD (50.9% vs 23.6%; p=0.003).

Table 2: Spirometric Parameters, GOLD Severity Classification and Exacerbation Frequency

Variable	S-COPD (n=55)	B-COPD (n=55)	p-value
Post-BD FEV_1 (% predicted), mean ± SD	46.8 ± 14.2	51.6 ± 13.7	0.049*
Post-BD FVC (% predicted), mean ± SD	61.3 ± 13.1	66.8 ± 12.4	0.027*
FEV_1/FVC ratio, mean ± SD	0.57 ± 0.09	0.60 ± 0.08	0.063
GOLD Grade I ($FEV_1 \geq 80\%$), n (%)	3 (5.5%)	6 (10.9%)	—
GOLD Grade II (FEV_1 50–79%), n (%)	16 (29.1%)	23 (41.8%)	—
GOLD Grade III (FEV_1 30–49%), n (%)	24 (43.6%)	20 (36.4%)	—
GOLD Grade IV ($FEV_1 < 30\%$), n (%)	12 (21.8%)	6 (10.9%)	—
Overall GOLD Grade distribution	—	—	0.038*
CAT Score, mean ± SD	22.7 ± 5.4	20.1 ± 5.8	0.019*
mMRC Dyspnoea Scale, mean ± SD	2.8 ± 0.9	2.5 ± 1.0	0.090
6-Minute Walk Distance (m), mean ± SD	286.4 ± 74.1	304.2 ± 68.3	0.187
Mean annual exacerbations ± SD	2.6 ± 1.2	1.8 ± 1.0	0.001*
0 exacerbations, n (%)	5 (9.1%)	11 (20.0%)	—
1–2 exacerbations, n (%)	27 (49.1%)	34 (61.8%)	—
≥ 3 exacerbations, n (%)	23 (41.8%)	10 (18.2%)	—
Overall exacerbation category	—	—	0.009*
Hospitalisation in last 12 months, n (%)	31 (56.4%)	18 (32.7%)	0.011*
SpO_2 at rest (%), mean ± SD	90.3 ± 4.6	91.7 ± 4.1	0.089
GOLD Group A, n (%)	7 (12.7%)	14 (25.5%)	—
GOLD Group B, n (%)	20 (36.4%)	28 (50.9%)	—
GOLD Group E, n (%)	28 (50.9%)	13 (23.6%)	—
Overall ABE distribution	—	—	0.003*

BD = Bronchodilator; FEV_1 = Forced Expiratory Volume in 1 second; FVC = Forced Vital Capacity; CAT = COPD Assessment Test; mMRC = Modified Medical Research Council; SpO_2 = Peripheral Oxygen Saturation; ABE = GOLD A/B/E classification. *p<0.05.

Radiological Findings: On plain chest radiography, the S-COPD group more often showed hyperinflation and barrel-chest deformity (69.1% vs 43.6%; p=0.007), hemidiaphragm flattening (65.5% vs 40.0%; p=0.007), and an enlarged retrosternal space (56.4% vs 34.5%; p=0.021). B-COPD patients, on the other hand, had more prominent bronchovascular markings (74.5% vs 52.7%; p=0.018). HRCT findings further differentiated the two groups. Centrilobular emphysema was the hallmark of S-COPD on CT (72.7% vs 27.3%; p<0.001), and panlobular emphysema was also more prevalent there (32.7% vs 10.9%; p=0.004).

The B-COPD CT profile looked quite different: bronchial wall thickening was found in nearly 70% of this group (69.1% vs 38.2%; p=0.001), peribronchial thickening in 41.8% (vs 16.4%; p=0.003), air trapping in 63.6% (vs 45.5%; p=0.041), and bronchiectasis in 30.9% (vs 12.7%; p=0.021). Fibrotic bands were also more common in B-COPD (27.3% vs 10.9%; p=0.026). Taking it all together, emphysema-predominant phenotype was seen in 67.3% of S-COPD versus just 21.8% of B-COPD (p<0.001), while airway disease predominance was found in 56.4% of B-COPD versus only 20.0% of S-COPD (p<0.001).

Table 3: Radiological Findings on Chest X-ray and High-Resolution CT Chest

Radiological Feature	S-COPD (n=55) n (%)	B-COPD (n=55) n (%)	p-value
Hyperinflation / Barrel chest	38 (69.1%)	24 (43.6%)	0.007*
Increased bronchovascular markings	29 (52.7%)	41 (74.5%)	0.018*
Flattened hemidiaphragm	36 (65.5%)	22 (40.0%)	0.007*
Enlarged retrosternal airspace	31 (56.4%)	19 (34.5%)	0.021*
Cardiomegaly	10 (18.2%)	18 (32.7%)	0.075
Centrilobular emphysema	40 (72.7%)	15 (27.3%)	<0.001*
Panlobular emphysema	18 (32.7%)	6 (10.9%)	0.004*
Paraseptal emphysema	14 (25.5%)	7 (12.7%)	0.087
Air trapping	25 (45.5%)	35 (63.6%)	0.041*
Bronchial wall thickening	21 (38.2%)	38 (69.1%)	0.001*
Peribronchial thickening	9 (16.4%)	23 (41.8%)	0.003*
Bronchiectasis	7 (12.7%)	17 (30.9%)	0.021*
Mosaic perfusion pattern	11 (20.0%)	20 (36.4%)	0.057
Fibrotic bands / Scarring	6 (10.9%)	15 (27.3%)	0.026*
Ground glass opacities	5 (9.1%)	8 (14.5%)	0.375
Pulmonary artery enlargement	14 (25.5%)	22 (40.0%)	0.099
Emphysema-predominant CT phenotype	37 (67.3%)	12 (21.8%)	<0.001*
Airway disease-predominant CT phenotype	11 (20.0%)	31 (56.4%)	<0.001*
Mixed CT phenotype	7 (12.7%)	12 (21.8%)	0.209

HRCT = High-Resolution Computed Tomography. *p<0.05. Chi-square test for all categorical comparisons.

Discussion

The findings from this study add to a growing body of evidence that tobacco-related and biomass-related COPD are not simply two versions of the same disease. Across clinical severity, exacerbation burden, spirometric performance, and CT phenotyping, the two groups behaved differently in ways that are consistent, biologically plausible, and clinically important.

The demographic profile we observed — predominantly male S-COPD patients and predominantly female B-COPD patients — was expected, but it is worth pausing on what it reflects. In India, men smoke tobacco at rates ten times higher than women, yet the male-to-female COPD ratio is only about 1.5:1 [8]. That asymmetry makes little sense unless women are acquiring COPD through a different route — namely, years of cooking over open biomass fires in poorly ventilated spaces. Our B-COPD group, where nearly three-quarters of patients were women from rural areas, exemplifies this pattern precisely. Singh et al. [15] observed a similar female predominance (70%) in a non-smoker COPD cohort from North India, where biomass exposure accounted for the majority of attributable risk.

On spirometry, the differences were statistically significant but perhaps not as large as one might expect — S-COPD patients had lower FEV_1% predicted (46.8% vs 51.6%; p=0.049), and a higher proportion clustered in GOLD Grades III–IV (65.4% vs 47.3%; p=0.038). CAT scores were also worse in S-COPD, pointing to a greater day-to-day symptom burden. This fits with data from De Paco-Matallana et al. [13] and others suggesting that tobacco smoke causes more aggressive spirometric decline over time.

What we found on exacerbation frequency was, in our view, one of the more clinically meaningful results. S-COPD patients had significantly more exacerbations per year (2.6 vs 1.8; p=0.001), and a much larger proportion — over 40% — had three or more acute episodes in the preceding 12 months. Hospitalisation rates followed the same pattern. These results might seem counterintuitive if one assumes that milder spirometric impairment in B-COPD translates to a quieter clinical course. But the literature has been fairly consistent on this point. The COPET study by Golpe et al. [16] specifically found tobacco-exposed patients were more likely to be frequent exacerbators, and the meta-analysis by Hu et al. [17] reported a lower exacerbation risk ratio in B-COPD (RR 0.74; 95% CI 0.59–0.94). Our GOLD Group E distribution — 50.9% in S-COPD versus 23.6% in B-COPD — reinforces this finding. The CT findings were, in some ways, the most visually compelling part of this study. Centrilobular emphysema — the classic consequence of tobacco-induced alveolar destruction radiating outward from terminal bronchioles — was nearly three times more prevalent in S-COPD (72.7% vs 27.3%; p<0.001). This is consistent with what Camp et al. [9] found when comparing tobacco versus wood-smoke COPD in Mexican women, and what Parada et al. [18] demonstrated using quantitative CT indices in a Brazilian cohort.

B-COPD, by contrast, showed a distinctly airway-centred radiological signature. Bronchial wall

thickening, peribronchial thickening, air trapping, and bronchiectasis were all significantly more prevalent. This is not surprising mechanistically — biomass smoke appears to cause lymphocyte-driven inflammation, basement membrane thickening, and small-airway fibrosis, as reviewed by Dutta et al. [10], producing exactly the pattern we observed. González-García et al. [19] reported similar findings from Colombian wood-smoke COPD patients, where bronchial wall thickening and dilatation were the dominant CT abnormalities. The higher rate of bronchiectasis in B-COPD in our study likely reflects recurrent cycles of airway inflammation and infection layered onto a structurally compromised, thickened bronchial wall — a process that spirometry alone would not capture. Pulmonary hypertension and cor pulmonale were significantly more common in our B-COPD group (47.3% and 41.8% respectively), which is a finding with real clinical weight. Despite less severe airflow obstruction, these patients appear to develop substantial pulmonary vascular disease — likely through a combination of chronic hypoxaemia, ventilation-perfusion mismatch from small-airway disease, and possibly direct vascular effects of combustion products. Parada et al. [18] have documented worse arterial oxygenation in biomass COPD relative to spirometrically comparable tobacco COPD patients. Salvi [5] highlighted the prevalence of cor pulmonale in rural Indian women with non-smoking COPD. If these vascular complications are not actively screened for, B-COPD patients could be systematically under-treated despite appearing less severe on standard measures. This study has notable strengths: rigorous single-exposure eligibility criteria, standardised spirometry, blinded radiological evaluation, and a cohort from an underrepresented region of India. Limitations include the cross-sectional design (which cannot establish causation), reliance on patient-reported exacerbation history, and a sample size that may not detect differences in less prevalent outcomes. The strict single-exposure criterion, while useful for phenotypic clarity, limits how far findings can be generalised to real-world patients — many of whom have mixed exposure histories. Future work should include prospective follow-up, objective exposure measurement, and larger samples to address these gaps.

Conclusion

This study makes a reasonably clear case that smoking-related and biomass-related COPD are distinct phenotypes — not just at the epidemiological level, but in how they present clinically, how they behave over time, and what they look like on imaging. Patients with tobacco-related COPD have worse spirometry, more frequent exacerbations, and more admissions; their CT scans are dominated by emphysema. Patients with biomass-related COPD, despite appearing less obstructed, carry a heavier burden of airway disease on imaging and are far more likely to develop pulmonary hypertension and cor pulmonale.

These distinctions matter for practice. B-COPD patients need active screening for pulmonary vascular disease; S-COPD patients need aggressive exacerbation prevention. And at a broader level, this data adds to the case for treating biomass fuel transition in rural Bihar — and across India — not as a future ambition but as an immediate public health priority.

Declarations

Conflict of Interest: None declared.

Source of Funding: Nil.

Ethical Approval: Approved by the Institutional Ethics Committee, Jawaharlal Nehru Medical College & Hospital, Bhagalpur. All procedures complied with the ethical standards of institutional and national research guidelines and the 1964 Helsinki Declaration and its later amendments.

Informed Consent: Written informed consent was obtained from all individual participants.

Author Contributions

RKV — Conceptualisation, methodology, data collection, statistical analysis, manuscript writing and corresponding authorship. AK — Data collection, literature review, clinical investigation. SP — Data collection, radiological data recording, critical revision. All authors approved the final manuscript.

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