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CT findings in biomass smoke-associated interstitial lung disease: A retrospective analysis

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ABSTRACT

Objectives: Household air pollution from biomass fuel combustion remains a major global health burden, affecting nearly 3 billion people worldwide. Although biomass smoke exposure is a recognized risk factor for chronic airway disease, its role in interstitial lung disease (ILD) is less well defined, and characteristic CT features have not been systematically described in patients without coexisting chronic obstructive pulmonary disease (COPD).

Material and Methods: We conducted a retrospective review of 23 patients with biomass smoke exposure and radiographic evidence of ILD, excluding those with alternative ILD etiologies or pre-existing COPD. CT scans were independently assessed by a thoracic radiologist and two pulmonologists for predominant patterns, distributions, and additional findings, with consensus classification. We report proportions with 95% confidence intervals (CIs) and performed exploratory Spearman correlation to assess associations between selected CT features.

Results: The cohort (mean age 71.7 years; all females; 96% born outside the United States) most commonly demonstrated reticulation (16/23, 70%; 95% CI, 49–84%), ground-glass opacities (GGO) (15/23, 65%; 95% CI, 45–81%), mosaic attenuation (15/23, 65%; 95% CI, 45–81%), and bronchial wall thickening (14/23, 61%; 95% CI, 41–78%). Abnormalities were typically patchy (18/23, 78%; 95% CI, 58–90%) and peribronchial (13/23, 57%; 95% CI, 37–74%), without consistent apical or basal predominance. Traction bronchiectasis occurred in 12/23 (52%; 95% CI, 33–71%) and lymphadenopathy in 9/23 (39%; 95% CI, 22–59%). Honeycombing (5/23, 22%; 95% CI, 10–42%), centrilobular emphysema (4/23, 17%; 95% CI, 7–37%), nodules (3/23, 13%; 95% CI, 5–32%), cysts (2/23, 9%; 95% CI, 2–27%), tree-in-bud opacities (2/23, 9%; 95% CI, 2–27%), and centrilobular nodularity (1/23, 4%; 95% CI, 1–21%) were uncommon. Exploratory Spearman analyses showed strong associations between mosaic attenuation and bronchial wall thickening ($p = 0.91$) and between peribronchial distribution and mosaic attenuation ($p = 0.83$), with additional positive correlations between mosaic attenuation and bronchiectasis ($p = 0.70$), peribronchial distribution and bronchial wall thickening ($p = 0.73$), bronchial wall thickening and bronchiectasis ($p = 0.77$), bronchiectasis and traction bronchiectasis ($p = 0.57$), and reticulation and traction bronchiectasis ($p = 0.69$). Exploratory Spearman correlation analyses showed strong associations between mosaic attenuation and bronchial wall thickening ($p = 0.91$) and between peribronchial distribution and mosaic attenuation ($p = 0.83$), with additional positive correlations between mosaic attenuation and bronchiectasis ($p = 0.70$), peribronchial distribution and bronchial wall thickening ($p = 0.73$), bronchial wall thickening and bronchiectasis ($p = 0.77$), bronchiectasis and traction bronchiectasis ($p = 0.57$), and reticulation and traction bronchiectasis ($p = 0.69$).

Conclusion: Biomass smoke-associated ILD demonstrates a distinctive CT pattern characterized by reticulation, GGOs, and mosaic attenuation with patchy and peribronchial distribution and no clear zonal predominance.

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Unlike hypersensitivity pneumonitis, it lacks centrilobular nodularity. Compared with idiopathic pulmonary fibrosis (IPF), it shows less extensive subpleural honeycombing, more peribronchial involvement, and no consistent basal predominance. These distinctions may improve recognition of biomass smoke-associated ILD in exposed patients. Further prospective studies are needed to validate these findings and clarify their prognostic significance.

Keywords: Biomass smoke, Computed tomography, Household air pollution, Interstitial lung disease, Pulmonary fibrosis

INTRODUCTION

Household air pollution from biomass fuel combustion remains a major global health burden, affecting nearly 3 billion people worldwide. Biomass fuels – including wood, animal dung, agricultural residues, and coal – are widely used for cooking, heating, and lighting in low- to middle-income countries where alternative clean energy sources are often unavailable or prohibitively expensive. In poorly ventilated settings, combustion of these fuels generates indoor air pollution with fine particulate matter concentrations exceeding acceptable air quality limits by more than 100-fold.^[1,2] In 2016, the World Health Organization estimated that 3.8 million deaths were attributable to household air pollution, accounting for approximately 7.7% of global mortality.^[3] Despite the scale of this public health crisis, 2.6 billion people worldwide still lacked access to clean cooking fuels as of 2019, with women and children in regions such as Asia, Africa, and Latin America disproportionately affected.^[4,5]

The adverse health effects of biomass smoke exposure are extensive and well-documented, including increased risks of respiratory infections, chronic obstructive pulmonary disease (COPD), cardiovascular disease, and lung cancer.^[6-10] Biomass smoke contains a complex mixture of toxic compounds – including particulate matter (PM_{2.5}), carbon monoxide, nitrogen oxides, aldehydes, and polycyclic aromatic hydrocarbons – that can induce inflammation, oxidative stress, and endothelial injury.^[11-13] This inflammatory cascade has been linked to adverse cardiac remodeling, pulmonary hypertension, and reduced birth weight in exposed populations.^[14-16] Long-term PM_{2.5} exposure has also been associated with cardiovascular disease.^[17] Ambient air pollution has been associated with reduced oxygen saturation,^[18] and recent updates on biomass and traffic pollution further support these associations.^[19] Short-term elevations in PM_{2.5} have also been linked to acute lower respiratory infection.^[20]

While biomass smoke is classically linked with airway disease and COPD,^[21-25] emerging evidence suggests that it may also contribute to the development of interstitial lung disease (ILD). However, the computed tomography (CT) imaging characteristics of biomass smoke-associated ILD remain poorly defined, and few studies have specifically examined this entity in patients without pre-existing COPD.

Identifying distinctive radiographic features could facilitate earlier diagnosis, reduce reliance on invasive procedures, and improve patient outcomes.

This study aims to characterize the CT imaging findings of biomass smoke-associated ILD and assess whether these patterns differ from those observed in other forms of ILD. A clearer understanding of these imaging characteristics may enhance diagnostic accuracy, guide clinical management, and inform public health strategies in populations at-risk.

MATERIAL AND METHODS

Patients

Patients were identified through a chart review of individuals evaluated in an academic pulmonology clinic who had both a documented history of biomass smoke exposure and radiographic evidence of ILD on chest CT. Exclusion criteria included an alternative identified cause of ILD, such as connective tissue disease, silicosis, or inhalation of environmental antigens associated with hypersensitivity pneumonitis (HP). Patients with a pre-existing diagnosis of COPD were also excluded to avoid confounding radiographic interpretation.

CT protocol and analysis

Chest CT examinations were performed on either a 64-slice (Somatom Sensation 64; Siemens, Erlangen, Germany) or a 128-slice Multidetector computed tomography (MDCT) scanner (Somatom Sensation AS+, Siemens; or Ingenuity Core 128, Philips Healthcare, Cleveland, OH, USA). Scans were obtained after a scout image to define the field of view, using standard parameters of 120 kVp, 100–200 mA, and 1–3 mm slice thickness, with reconstruction at 1–3 mm intervals in the mediastinal window. Images were reconstructed with standard lung (high-spatial-frequency) and mediastinal kernels; evaluations of interstitial and airway findings were performed on lung windows. Images were reconstructed on vendor workstations and archived in the institutional Picture archiving and communication system (PACS) (Centricity 4.0; GE Medical Systems, Mount Prospect, IL, USA).

Chest CT scans were independently reviewed by a thoracic radiologist and two pulmonologists with >10 years' experience

in ILD, with consensus achieved on the predominant imaging patterns. Findings were categorized according to American Thoracic Society/European Respiratory Society criteria,^[26] including the presence of ground-glass opacities (GGOs), reticulation, honeycombing, cysts, centrilobular emphysema, and nodules. The distribution of abnormalities (peripheral, peribronchial, patchy, or diffuse) and the presence of additional features such as bronchial wall thickening, bronchiectasis, mosaic attenuation, pleural thickening, and lymphadenopathy were also recorded.

Statistical analysis

This was a descriptive case series. Continuous variables are summarized as mean $\pm$ standard deviation or median (range), as appropriate. Categorical variables are reported as counts and percentages with 95% confidence intervals (CIs) for key imaging findings (Wilson score method). Exploratory associations between selected CT features were assessed using Spearman's rank correlation (ρ); binary CT variables were coded as 1 = present and 0 = absent. Given the exploratory design and sample size ($n = 23$), these association analyses were interpreted descriptively.

For these exploratory analyses, CT features were selected before reflecting airway-centered abnormalities (mosaic attenuation, bronchial wall thickening, bronchiectasis) and fibrotic remodeling (reticulation, traction bronchiectasis).

RESULTS

Twenty-three patients with a documented history of biomass smoke exposure and radiographic evidence of ILD were included in the analysis. The mean age was 71.7 years, and all patients were female. Most participants (96%) were born outside of the United States, predominantly in Latin America (91%) and the Philippines (4%). The median duration of biomass smoke exposure was 30 years (range, 10–80 years), with a median of 6 h of exposure per day (range, 3–10 h/day) [Table 1].

The most frequent CT patterns observed were reticulation (16/23, 70%; 95% CI, 49–84%) and GGO (15/23, 65%; 95% CI, 45–81%), followed by honeycombing (5/23, 22%; 95% CI, 10–42%), centrilobular emphysema (4/23, 17%; 95% CI, 7–37%), nodules (3/23, 13%; 95% CI, 5–32%), cysts (2/23, 9%; 95% CI, 2–27%), and consolidation (1/23, 4%; 95% CI, 1–21%) [Table 2 and Figure 1]. Among nodular patterns, tree-in-bud nodules were observed in 2/23 (9%; 95% CI, 2–27%) and centrilobular nodules in 1/23 (4%; 95% CI, 1–21%), whereas perilymphatic and random nodules were not seen.

The predominant distribution of CT findings was patchy (18/23, 78%; 95% CI, 58–90%) and peribronchial (13/23,

Table 1: Baseline characteristics of the study population ($n=23$).

Characteristic	n (%) or value
Sex	
Female	23 (100)
Age, years	
Mean $\pm$ SD	71.7 $\pm$ 15
Country/region of origin	
Non-US	22 (96)
Latin America	21 (91)
Mexico	8 (36)
El Salvador	8 (36)
Guatemala	3 (13)
Honduras	1 (4)
Colombia	1 (4)
Philippines	1 (4)
Occupation	
Homemaker	18 (78)
Cook	2 (9)
Housekeeper	1 (4)
Florist	1 (4)
Hypnotherapist	1 (4)
Other indoor exposures	
Passive tobacco smoking	4 (17)
Active tobacco smoking	2 (9)
Coal	1 (4)
Petroleum	1 (4)
Duration of biomass smoke exposure	
Hours per day, median (range)	6 (3–10)
Total years, median (range)	30 (10–80)
Values are n (%) unless otherwise indicated. Percentages use $n=23$ as the denominator. SD: Standard deviation	

57%; 95% CI, 37–74%), with less frequent peripheral (8/23, 35%; 95% CI, 19–55%) and diffuse (4/23, 17%; 95% CI, 7–37%) involvement; subpleural sparing was rare (1/23, 4%; 95% CI, 1–21%). With respect to zonal predominance, 10/23 (43%; 95% CI, 26–63%) had no obvious gradient, while basal (5/23, 22%; 95% CI, 10–42%), apical (4/23, 17%; 95% CI, 7–37%), and apicobasal gradients (4/23, 17%; 95% CI, 7–37%) were each observed in smaller proportions [Table 2 and Figure 2].

Bronchial findings included bronchial wall thickening (14/23, 61%; 95% CI, 41–78%), traction bronchiectasis (12/23, 52%; 95% CI, 33–71%), and bronchiectasis (11/23, 48%; 95% CI, 29–67%). Pleural abnormalities were infrequent, limited to pleural thickening (3/23, 13%; 95% CI, 5–32%), with no effusions noted (0/23, 0%; 95% CI, 0–14%). Other findings included mosaic attenuation

Table 2: Chest CT findings ($n=23$).

CT finding	<i>n</i> (%)	95% CI (%)
Pattern		
Reticulation	16 (70)	49–84
Ground-glass opacities	15 (65)	45–81
Honeycombing	5 (22)	10–42
Centrilobular emphysema	4 (17)	7–37
Nodule	3 (13)	5–32
Cyst	2 (9)	2–27
Consolidation	1 (4)	1–21
Nodular type		
Tree-in-bud	2 (9)	2–27
Centrilobular	1 (4)	1–21
Perilymphatic	0 (0)	0–14
Random	0 (0)	0–14
Distribution		
Patchy	18 (78)	58–90
Peribronchial	13 (57)	37–74
Peripheral	8 (35)	19–55
Diffuse	4 (17)	7–37
Subpleural sparing	1 (4)	1–21
Gradient		
No obvious gradient	10 (43)	26–63
Basal predominance	5 (22)	10–42
Apical predominance	4 (17)	7–37
Apicobasal gradient	4 (17)	7–37
Bronchial findings		
Bronchial wall thickening	14 (61)	41–78
Traction bronchiectasis	12 (52)	33–71
Bronchiectasis	11 (48)	29–67
Pleural findings		
Pleural thickening	3 (13)	5–32
Pleural effusion	0 (0)	0–14
Other findings		
Mosaic attenuation	15 (65)	45–81
Lymphadenopathy	9 (39)	22–59

Values are *n* (%). Percentages use $n=23$ as the denominator. 95% CIs are Wilson score intervals. CT: Computed tomography, CI: Confidence interval

(15/23, 65%; 95% CI, 45–81%) and lymphadenopathy (9/23, 39%; 95% CI, 22–59%).

In exploratory Spearman analyses of CT findings coded as present/absent, mosaic attenuation demonstrated a strong positive correlation with bronchial wall thickening ($\rho = 0.91$) and with peribronchial distribution ($\rho = 0.83$) and a moderate correlation with bronchiectasis ($\rho = 0.70$).

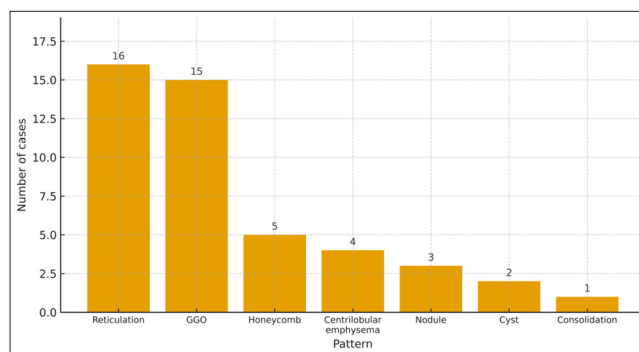


Figure 1: Cohort of 23 patients with biomass smoke-associated interstitial lung disease. Chest CT, lungs; bar chart shows the frequency of CT imaging patterns: reticulation, ground-glass opacity, honeycombing, centrilobular emphysema, nodules, cysts, and consolidation. Numbers above bars indicate the number of cases for each pattern (patterns are not mutually exclusive). CT: Computed tomography.

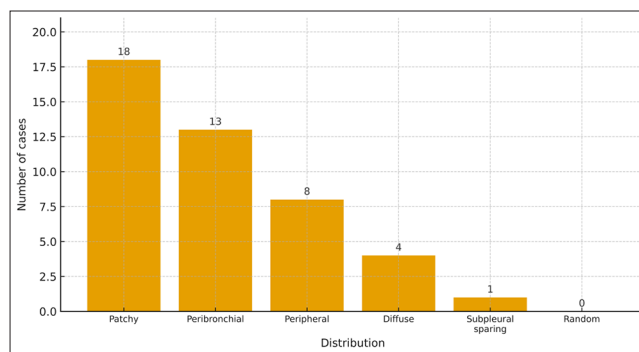


Figure 2: Cohort of 23 patients with biomass smoke-associated interstitial lung disease. Chest CT, lungs; bar chart shows the distribution of imaging abnormalities (patchy, peribronchial, peripheral, diffuse, subpleural sparing, and random). Numbers above bars indicate the number of cases for each distribution category (categories are not mutually exclusive). CT: Computed tomography.

Peribronchial distribution was also positively correlated with bronchial wall thickening ($\rho = 0.73$). Among airway and fibrotic features, bronchial wall thickening correlated with bronchiectasis ($\rho = 0.77$), and bronchiectasis correlated with traction bronchiectasis ($\rho = 0.57$). Reticulation demonstrated a moderate correlation with traction bronchiectasis ($\rho = 0.69$), supporting overlap between fibrotic remodeling and airway distortion in this cohort [Table 3].

DISCUSSION

The CT findings in this cohort suggest that biomass smoke-associated ILD exhibits a distinctive radiographic profile characterized by reticulation (16/23, 70%; 95% CI, 49–84%), GGOs (15/23, 65%; 95% CI, 45–81%), mosaic attenuation (15/23, 65%; 95% CI, 45–81%), and bronchial wall thickening

(14/23, 61%; 95% CI, 41–78%) [Figure 3]. These features differ from the centrilobular nodularity characteristic of HP and the dense fibrosis typical of IPF and likely reflect a combination of airway inflammation, early fibrosis, and small airway disease resulting from prolonged biomass smoke exposure.

The distribution of findings was most often patchy (78%) and peribronchial (57%), with less frequent peripheral (35%) and diffuse (17%) involvement. Importantly, no clear zonal predominance was seen: 43% showed no gradient, while basal (22%), apical (17%), and apicobasal (17%) predominance were each observed in smaller proportions [Figure 4]. This lack of a dominant gradient contrasts with the basal predominance of IPF and the upper-lobe involvement of HP, and may reflect the diffuse, persistent nature of biomass smoke exposure rather than the gravitational or ventilatory influences that shape the distribution of other ILDs.

The observation of both traction bronchiectasis (52%) and bronchiectasis not clearly attributable to fibrosis (48%) is particularly noteworthy. In IPF, traction bronchiectasis

usually arises as a secondary marker of advanced architectural distortion and dense subpleural fibrosis, typically in association with extensive honeycombing. In this cohort, however, traction bronchiectasis occurred in the setting of patchier fibrotic changes and more frequent peribronchial involvement. This suggests that airway-centered injury and remodeling may play a more central pathogenic role in biomass smoke-associated ILD. The coexistence of traction bronchiectasis and primary bronchiectasis points toward a dual injury mechanism involving both direct airway-centered damage with secondary airway dilation and fibrosis-related traction changes. This pattern aligns with the hypothesis that biomass smoke exposure causes chronic small airway inflammation and fibrosis that distort and dilate the bronchi, rather than traction bronchiectasis serving exclusively as a marker of late-stage fibrosis. The presence of both airway-centered and traction-related airway changes may therefore provide an additional radiographic clue distinguishing biomass smoke-associated ILD from other fibrotic lung diseases.

Honeycombing was present in only 22% of patients, substantially lower than what is typically seen in IPF where honeycombing is extensive, basal-predominant, and subpleural. In this cohort, honeycombing lacked a consistent zonal predominance and was less confluent, often occurring in the context of patchier and peribronchial fibrotic changes. These distinctions reinforce the concept that biomass smoke-associated ILD involves less dense subpleural fibrosis compared to IPF, and that its fibrotic manifestations are more airway-centered.

Nodularity was uncommon in this cohort, with tree-in-bud nodules observed in 9% of cases and centrilobular nodules in only 4%. This contrasts sharply with HP, where centrilobular nodularity is a hallmark feature.^[27,28] Mosaic attenuation, on the other hand, was observed in nearly two-thirds of patients. In HP, mosaic attenuation is typically attributed

Table 3: Spearman correlations between selected CT features (n=23).

CT feature pair	Spearman ρ
Mosaic attenuation↔Bronchial wall thickening	0.91
Mosaic attenuation↔Bronchiectasis	0.70
Peribronchial distribution↔Bronchial wall thickening	0.73
Peribronchial distribution↔Mosaic attenuation	0.83
Bronchial wall thickening↔Bronchiectasis	0.77
Bronchiectasis↔Traction bronchiectasis	0.57
Reticulation↔Traction bronchiectasis	0.69

Spearman ρ values are presented as effect sizes; P-values are not shown

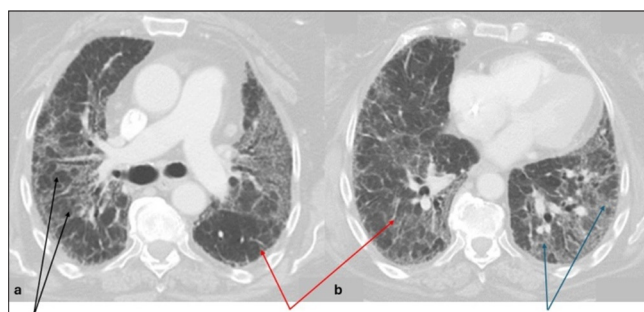


Figure 3: 72-year-old woman with chronic dry cough and dyspnea on exertion due to biomass smoke-associated interstitial lung disease. (a) Contrast-enhanced CT of the chest, axial lung-window image shows patchy peribronchovascular-predominant reticulation (red arrow) and areas of mosaic attenuation (black arrows). (b) Contrast-enhanced CT of the chest, axial lung-window image shows reticulation (red arrow) and ground-glass opacities (blue arrows). CT: Computed tomography.

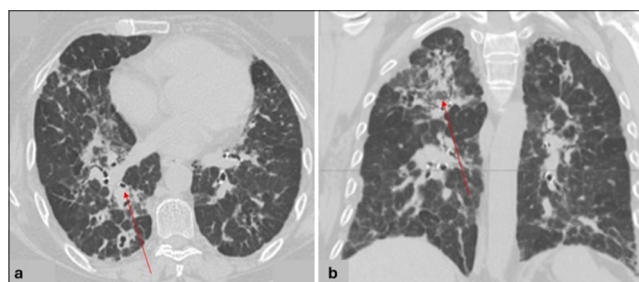


Figure 4: 78-year-old woman with insidious onset of dyspnea on exertion due to biomass smoke-associated interstitial lung disease. (a) CT of the chest, axial lung-window image shows patchy peribronchovascular predominant involvement (red arrow). (b) CT of the chest, coronal lung-window image, corroborates the patchy peribronchovascular pattern (red arrow). CT: Computed tomography.

to widespread small airway obstruction with prominent air trapping; in biomass smoke-associated ILD, it is more likely a combination of small airway disease and heterogeneous fibrosis in the absence of nodularity. Together, the low prevalence of nodularity and the distinct pattern of mosaic attenuation further distinguish this entity from HP.

In addition to the descriptive CT profile, exploratory correlation analysis provides further support for a mixed airway- and fibrosis-centered phenotype in biomass smoke-associated ILD. Mosaic attenuation correlated strongly with bronchial wall thickening and peribronchial distribution, and was also associated with bronchiectasis, suggesting that small airways dysfunction and airway remodeling frequently co-occur with the interstitial abnormalities observed in this cohort. The correlation between reticulation and traction bronchiectasis further links fibrotic remodeling with airway distortion. These associations should be interpreted descriptively given the sample size and exploratory design, but they are consistent with the hypothesis that chronic biomass smoke exposure produces overlapping small airway injury and patchy fibrotic change rather than a purely subpleural, basal-predominant pattern.

Lymphadenopathy was seen in 39% of patients. This is relatively uncommon in both IPF and chronic HP, suggesting a unique contribution of biomass smoke exposure to persistent airway inflammation and antigenic stimulation.

The relatively high frequency of lymphadenopathy in this cohort provides an additional distinguishing feature when considering the differential diagnosis of ILD.

When contrasting biomass smoke-associated ILD with HP and IPF, several key differences emerge [Table 4]. Unlike HP, biomass smoke-associated ILD in this study lacked centrilobular nodularity, a hallmark feature of HP. Although mosaic attenuation was common in both diseases, its likely origin differs: in HP, mosaic attenuation typically results from widespread small airway obstruction with accompanying air trapping, whereas in biomass smoke-associated ILD, it may represent a combination of small airway disease and heterogeneous fibrotic change without the classic HP nodular pattern. Compared with IPF, biomass smoke-associated ILD demonstrated less dense fibrosis and substantially less subpleural honeycombing. In IPF, honeycombing is usually extensive, basal-predominant, and subpleural, whereas in biomass smoke-associated ILD, honeycombing was present in only 22% of cases, lacked a basal predominance, and was often less confluent. Similarly, while traction bronchiectasis and bronchiolectasis are common in both diseases, in IPF, they tend to occur in the setting of advanced, dense fibrosis with marked architectural distortion; in biomass smoke-associated ILD, they were observed alongside patchier fibrotic changes and more frequent peribronchial involvement.

Table 4: Comparative CT and clinical features across ILDs.

Feature	Biomass smoke-associated ILD	Hypersensitivity pneumonitis (HP)	Idiopathic pulmonary fibrosis (IPF)
Common CT patterns	Reticulation, GGOs, mosaic attenuation, bronchial wall thickening	Centrilobular nodules, GGOs, mosaic attenuation, air trapping	Reticulation, dense fibrosis, subpleural honeycombing
Centrilobular nodularity	Absent	Common, often with ill-defined nodules	Absent
Mosaic attenuation	Common; likely from mixed small airway disease and patchy fibrosis	Common; primarily from small airway obstruction with air trapping	Rare or absent
Honeycombing	Present in a minority of cases (22%); usually limited, non-basal predominant, less confluent	Uncommon; if present, usually reflects chronic/fibrotic HP	Hallmark feature; basal-predominant, extensive, subpleural
Traction bronchiectasis	Common (52%); occurs with patchy fibrosis and peribronchial involvement	May be present in chronic HP; associated with upper/mid-lobe fibrosis	Common; occurs with advanced dense fibrosis and architectural distortion
Distribution	Patchy (78%), peribronchial (57%), peripheral (35%); no clear apicobasal gradient	Often upper/mid-lobe predominant; peribronchovascular or diffuse	Basal-predominant, subpleural
Tree-in-bud nodularity	Rare (9%)	Common in subacute HP	Absent
Subpleural sparing	Rare (4%)	Can be seen in some non-fibrotic HP cases	Absent
Lymphadenopathy	Fairly common (39%)	May occur	Rare
Typical exposure history	Prolonged biomass smoke exposure (cooking/heating)	Repeated inhalation of environmental antigens	Often idiopathic; may have a history of gastroesophageal reflux or smoking

GGO: Ground-glass opacity, ILD: Interstitial lung disease, CT: Computed Tomography

Taken together, these radiographic distinctions, including the coexistence of bronchiectasis and traction bronchiectasis, the relative paucity of subpleural honeycombing, the peribronchial distribution, the absence of a basal-predominant gradient, the low prevalence of nodularity, and the relatively high frequency of lymphadenopathy, may help narrow the differential diagnosis in patients with ILD and compatible exposure histories.

The demographic profile of this cohort, which was predominantly older, female, and foreign-born, underscores the global burden of household air pollution as a cause of ILD. Patients in this population may present later in their disease course due to delayed recognition and limited access to healthcare, which further complicates both diagnosis and management.

This study has several important limitations. First, its retrospective design introduces the potential for selection bias and limits the ability to assess temporal relationships between exposure and disease progression. Second, the relatively small sample size reduces the generalizability of our findings and restricts subgroup analyses, such as evaluating potential sex- or age-related differences in radiographic patterns. Third, pathological confirmation was not available, which limits our ability to definitively exclude other ILD with overlapping features. In addition, potential confounders such as smoking history, occupational exposures, or coexisting airway disease were not uniformly assessed, which may have influenced the observed CT patterns. Finally, the absence of longitudinal imaging follow-up prevented assessment of disease progression or radiographic response to therapy. Future prospective studies with larger, more diverse patient populations, ideally incorporating detailed exposure histories, histopathologic correlation, and serial imaging, are needed to validate these findings and to explore differences in natural history, treatment response, and long-term outcomes.

CONCLUSION

This study provides preliminary evidence that biomass smoke-associated ILD exhibits a distinctive CT profile that differs meaningfully from both HP and IPF. Common features included reticulation, GGOs, mosaic attenuation, and bronchial wall thickening, typically with a patchy and peribronchial distribution and without a consistent apicobasal gradient. Unlike HP, biomass smoke-associated ILD was characterized by an absence of centrilobular nodularity despite frequent mosaic attenuation, suggesting a different underlying mechanism related to chronic small airway injury and heterogeneous fibrosis rather than immune-mediated bronchiolitis. Compared with IPF, biomass smoke-associated ILD demonstrated less extensive and less confluent subpleural honeycombing, greater peribronchial involvement, and no clear basal

predominance. The coexistence of both bronchiectasis and traction bronchiectasis, as well as the relatively frequent presence of lymphadenopathy, further supports the concept of a mixed airway- and fibrosis-centered injury pattern that distinguishes this entity from other fibrotic lung diseases. Exploratory Spearman correlations were consistent with this mixed pattern, demonstrating co-occurrence of mosaic attenuation with airway abnormalities and an association between reticulation and traction bronchiectasis.

Given the significant global health burden of biomass smoke exposure, recognition of these radiographic distinctions may aid earlier diagnosis and more accurate differentiation of biomass smoke-associated ILD from other ILD etiologies, potentially reducing the need for invasive testing in patients with compatible exposure histories. Larger, prospective studies with more diverse patient populations are needed to confirm these findings, assess prognostic implications, identify biomarkers of disease progression, and refine diagnostic criteria for this underrecognized condition.

Ethical approval: The institutional review board has waived ethical approval for this study as it is a retrospective analysis.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given consent for their images and other clinical information to be reported in the journal. The patient understands that the patient's names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Use of artificial intelligence (AI)-assisted technology for manuscript preparation: The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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