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Identification of Interstitial Lung Diseases in Smokers vs Non-Smokers Using HRCT

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ABSTRACT

Background: *Interstitial lung diseases (ILDs) are a diverse group of pulmonary disorders characterized by inflammation and fibrosis of the lung interstitium, often leading to irreversible respiratory impairment. Cigarette smoking is a well-established environmental risk factor implicated in the initiation and progression of ILD. High-resolution computed tomography (HRCT) plays a pivotal role in the early detection and classification of ILD patterns, offering diagnostic precision that can guide timely intervention. However, data comparing HRCT patterns of ILD in smokers versus non-smokers, particularly in South Asian populations, remain limited.* ***Objective****: To identify and compare HRCT findings of interstitial lung diseases in smokers and non-smokers and to evaluate the association between smoking status and ILD prevalence.* ***Methods****: This cross-sectional observational study was conducted at Hameed Latif Hospital, Lahore, from January to December 2023. A total of 72 adults (36 smokers, 36 non-smokers) aged 18–80 years underwent HRCT chest imaging. HRCT scans were interpreted independently by two radiologists blinded to smoking status. ILD was defined radiologically using ATS/ERS classification criteria. Associations between smoking, demographic variables, and ILD presence were analyzed using chi-square tests, with effect sizes (Cramer's V) and p-values reported; $p < 0.05$ was considered significant.* ***Results****: ILD was detected in 42 participants (58.3%), with a significantly higher prevalence among smokers (88.9%) than non-smokers (27.8%) ($p < 0.001$, Cramer's $V = 0.60$). The most frequent HRCT pattern was Usual Interstitial Pneumonia (UIP) (12.5%), followed by Non-Specific Interstitial Pneumonia (NSIP) (8.3%) and Desquamative Interstitial Pneumonia (DIP) (5.6%), all exclusively observed in smokers. Age and gender showed no significant association with ILD occurrence ($p = 0.143$ and $p = 0.733$, respectively).* ***Conclusion****: Smoking is strongly associated with ILD presence and fibrotic HRCT patterns, particularly UIP and DIP, indicating its role in pulmonary parenchymal injury and remodeling. HRCT serves as an effective tool for early ILD detection, emphasizing the need for routine imaging surveillance and smoking cessation strategies in high-risk individuals.*

Keywords
Interstitial Lung Disease, HRCT, Smoking, Usual Interstitial Pneumonia, Pulmonary Fibrosis, Desquamative Interstitial Pneumonia

INTRODUCTION

Interstitial lung diseases (ILDs) comprise a heterogeneous group of diffuse parenchymal disorders characterized by inflammatory and fibrotic remodeling of the alveolar–interstitial unit, leading to exertional dyspnea, cough, impaired gas exchange, and progressive respiratory failure (1). Despite advances in phenotyping and antifibrotic therapy, the global clinical burden of ILD remains substantial, with significant morbidity, premature mortality, and high healthcare utilization (2). Early and accurate delineation of ILD patterns is essential because prognosis and treatment selection vary markedly across entities such as usual interstitial pneumonia (UIP), nonspecific interstitial pneumonia (NSIP), hypersensitivity pneumonitis (HP), and smoking-related interstitial pneumonias including respiratory bronchiolitis–interstitial lung disease (RB-ILD) and desquamative interstitial pneumonia (DIP) (1). High-resolution computed tomography (HRCT) is central to contemporary diagnostic algorithms, providing pattern-based, noninvasive stratification that can obviate surgical lung biopsy in a large proportion of cases when integrated with clinical context and multidisciplinary assessment (3).

Cigarette smoking is among the most frequently implicated exposures in ILD pathogenesis, associated not only with classic smoking-related phenotypes (RB-ILD, DIP) but also with emphysema–fibrosis overlap and potential modification of the trajectory of idiopathic pulmonary fibrosis (IPF) (4). Proposed mechanisms include persistent epithelial injury, macrophage-driven inflammation, oxidative stress, and aberrant repair culminating in fibroblast activation and architectural distortion of the lung parenchyma (5). Clinically, smoking may blur radiologic boundaries by introducing concomitant small-airway disease and emphysema, complicating discrimination from other causes of diffuse lung abnormalities, including connective tissue disease–related ILD and chronic HP (6). In low- and middle-income settings, where tuberculosis (TB) and post-infectious scarring are prevalent, the interpretive challenge intensifies, and context-specific evidence is needed to guide imaging-led triage and referral pathways (2).

Within this landscape, a focused evaluation of HRCT patterns in smokers compared with non-smokers presenting for chest imaging can clarify how tobacco exposure shifts the probability of ILD, and the distribution of radiologic phenotypes encountered in routine practice (3,4). Such knowledge directly informs pretest probability estimates, optimizes diagnostic sequencing, and may identify high-risk subgroups who warrant expedited multidisciplinary review and antifibrotic consideration (2,3). Accordingly, we investigated adult patients undergoing HRCT at a tertiary center to quantify the association between smoking status and ILD presence and to characterize the spectrum of HRCT patterns across smokers versus non-smokers. We hypothesized that smoking is independently associated with higher ILD prevalence on HRCT and with greater representation of smoking-related and fibrotic patterns such as UIP and RB-ILD/DIP among smokers relative to non-smokers (4,5).

MATERIAL AND METHODS

This cross-sectional observational study was conducted in the Department of Radiology at Hameed Latif Hospital, Lahore, Pakistan, to evaluate the association between smoking status and interstitial lung disease (ILD) using high-resolution computed tomography (HRCT). The study period spanned from January to December 2023, during which adult patients referred for HRCT due to respiratory complaints or suspicion of pulmonary pathology were prospectively enrolled after informed consent. The investigation followed the ethical principles of the Declaration of Helsinki and received institutional approval from the hospital's research ethics committee (approval number: HLH/RES/23/ILD).

Participants were recruited consecutively using a non-probability purposive sampling approach to ensure adequate representation of both smokers and non-smokers. Eligibility criteria included adults aged 18 to 80 years of either sex who underwent HRCT of the chest for evaluation of chronic cough, dyspnea, or suspected interstitial pathology. Exclusion criteria comprised patients with known primary or metastatic lung malignancy, traumatic lung injury, congenital lung disease, pregnancy, or those unwilling to provide informed consent. Smoking status was categorized as "smoker" for individuals with a self-reported or documented history of daily smoking of any duration and "non-smoker" for those with no history of tobacco exposure. Each participant's demographic and clinical information, including age, gender, and relevant history, was recorded on a structured data sheet at the time of imaging.

All HRCT scans were performed using a multi-detector computed tomography system (Siemens SOMATOM Definition AS, Germany). Scanning parameters included 1-mm slice thickness, high spatial resolution reconstruction kernel, and full inspiratory acquisition in the supine position. HRCT images were interpreted independently by two consultant radiologists with more than five years of thoracic imaging experience, blinded to participants' smoking status. Any disagreement was resolved by consensus. Radiological findings were classified according to internationally accepted criteria from the American Thoracic Society and the European Respiratory Society for HRCT pattern recognition in ILD, including usual interstitial pneumonia (UIP), nonspecific interstitial pneumonia (NSIP), desquamative interstitial pneumonia (DIP), hypersensitivity pneumonitis (HP), and respiratory bronchiolitis–associated ILD (RB-ILD) (7). Additional non-fibrotic pathologies such as post-tuberculosis fibrosis, bronchiectasis, pneumonia, and emphysema were also documented.

The primary outcome variable was ILD presence, defined as any radiologically confirmed fibrotic or interstitial pattern on HRCT consistent with ILD criteria. Independent variables included smoking status, age, sex, and specific HRCT findings. To mitigate selection bias, consecutive sampling was maintained, and the radiologists' blinding to smoking history minimized detection bias. Confounding by age and sex was evaluated using stratified analyses. Missing data were handled through pairwise deletion since the proportion of missing entries was <5%.

A minimum sample size of 72 was estimated using a two-sided chi-square test, assuming a power of 80%, a significance level of 0.05, and an expected difference in ILD prevalence of 40% between smokers and non-smokers based on previous reports (8). Statistical analysis was performed using IBM SPSS Statistics version 24.0. Descriptive statistics (mean, standard deviation, frequency, and percentage) summarized demographic and imaging data. Categorical associations were examined using the chi-square test or Fisher's exact test where applicable. A p-value <0.05 was considered statistically significant. Effect sizes (Cramer's V) were calculated to quantify the strength of associations. All analyses were verified by an independent biostatistician to ensure statistical integrity and reproducibility. To ensure data integrity, all records were anonymized and stored in password-protected databases accessible only to the research team. The analytical process followed standardized coding and double-entry verification. Quality control included cross-validation of randomly selected imaging reports and consistency checks across statistical outputs to confirm reproducibility and internal validity of findings (9).

RESULTS

Among the 72 participants evaluated, the mean age was 54.2 ± 13.8 years, ranging from 20 to 77 years. The majority of patients were male (72.2%), with an equal proportion of smokers (36, 50.0%) and non-smokers (36, 50.0%). ILD was detected in 42 participants (58.3%), whereas 30 participants (41.7%) had no interstitial changes on HRCT. Age and gender were not significantly associated with ILD presence ($p = 0.143$ and $p = 0.733$, respectively), suggesting that smoking status rather than demographic variables predominantly influenced ILD distribution.

A strong and statistically significant association was found between smoking and ILD occurrence ($\chi^2 = 25.6$, $p < 0.001$, Cramer's $V = 0.60$), indicating a large effect size. Among smokers, 32 (88.9%) demonstrated ILD, compared with only 10 (27.8%) among non-smokers. Conversely, 26 non-smokers (72.2%) showed no radiological evidence of ILD compared with only 4 (11.1%) smokers, underscoring the marked disparity in HRCT findings between these groups.

HRCT evaluation revealed a predominance of fibrotic patterns among ILD-positive participants. The most frequent finding was Usual Interstitial Pneumonia (UIP) pattern, identified in 9 patients (12.5%), all ILD-positive ($p < 0.001$). Non-Specific Interstitial Pneumonia (NSIP) and Post-TB Fibrosis each accounted for 8.3% of cases, followed by Desquamative Interstitial Pneumonia (DIP) (5.6%) and Respiratory Bronchiolitis–ILD (4.2%). These findings were exclusively observed among ILD-positive individuals. Non-fibrotic abnormalities such as small airway disease (8.3%), aspiration pneumonia, or healed granulomatous infection occurred predominantly among ILD-negative participants ($p = 0.004$).

Age stratification showed that ILD prevalence increased with age, peaking in the 41–60 and 61–80-year groups (65.7% and 64.0%, respectively), though the difference was not statistically significant ($p = 0.143$). No significant gender-based disparity was noted in ILD prevalence ($p = 0.733$). Overall, the findings demonstrate that smoking is a major determinant of ILD occurrence and HRCT pattern distribution, with UIP and related fibrotic subtypes being most frequently associated with tobacco exposure. The analysis of the 72 participants revealed a distinct distribution pattern of demographic and radiological variables. The mean age of participants was 54.2 years (SD ±13.8), with the highest proportion belonging to the 41–60-year group (48.6%). ILD was detected in 58.3% of all cases. Among the 12 participants aged 20–40 years, only 3 (25.0%) showed ILD

findings on HRCT, compared with 23 (65.7%) in the 41–60-year group and 16 (64.0%) in the 61–80-year group. However, this increasing trend with age was not statistically significant (p = 0.143). The male-to-female ratio was 2.6:1, and although ILD appeared more frequent in males (61.5%) than females (50.0%), the difference did not reach statistical significance (p = 0.733).

A marked disparity emerged between smokers and non-smokers regarding ILD presence. As shown in Table 2, ILD was present in 32 smokers (88.9%) compared with 10 non-smokers (27.8%), while 26 non-smokers (72.2%) showed no ILD versus only 4 smokers (11.1%). The chi-square test confirmed a statistically significant relationship between smoking and ILD (p < 0.001), with a large effect size (Cramer's V = 0.60), indicating a strong association between tobacco exposure and ILD development. These findings emphasize the powerful influence of smoking on pulmonary interstitial changes visible on HRCT.

Table 1. Demographic Characteristics of Study Participants (N = 72)

Variable	Category	Frequency (n)	Percentage (%)	p-value
Age (years)	20–40	12	16.7	0.143
	41–60	35	48.6	
	61–80	25	34.7	
Gender	Male	52	72.2	0.733
	Female	20	27.8	
Smoking Status	Smokers	36	50.0	—
	Non-Smokers	36	50.0	—
ILD Presence	Yes	42	58.3	—
	No	30	41.7	—

Table 2. Association Between Smoking Status and ILD Presence

Smoking Status	ILD Present (n, %)	ILD Absent (n, %)	Total	p-value	Effect Size (Cramer's V)
Smokers	32 (88.9%)	4 (11.1%)	36	<0.001	0.60
Non-Smokers	10 (27.8%)	26 (72.2%)	36		
Total	42 (58.3%)	30 (41.7%)	72		

Table 3. Distribution of HRCT Findings Among All Participants and Their Association with ILD

HRCT Pattern	Total (n, %)	ILD Present (n, %)	ILD Absent (n, %)	p-value	Odds Ratio (95% CI)
Usual Interstitial Pneumonia (UIP)	9 (12.5)	9 (21.4)	0 (0.0)	<0.001	—
Non-Specific Interstitial Pneumonia (NSIP)	6 (8.3)	6 (14.3)	0 (0.0)	<0.001	—
Desquamative Interstitial Pneumonia (DIP)	4 (5.6)	4 (9.5)	0 (0.0)	<0.001	—
Respiratory Bronchiolitis–ILD (RB-ILD)	3 (4.2)	3 (7.1)	0 (0.0)	0.008	—
Post-TB Fibrosis	6 (8.3)	6 (14.3)	0 (0.0)	<0.001	—
Small Airway Disease	6 (8.3)	0 (0.0)	6 (20.0)	0.004	—
Other Patterns (combined)*	38 (52.8)	14 (33.3)	24 (80.0)	—	—
Total	72 (100)	42 (58.3)	30 (41.7)	—	—

***Includes bronchopneumonia, healed granulomatous disease, pleural effusion, emphysema, and other non-fibrotic pathologies.**

Table 4. Age-Wise Distribution of ILD Among Participants

Age Group (years)	ILD Present (n, %)	ILD Absent (n, %)	Total (n)	p-value
20–40	3 (25.0)	9 (75.0)	12	0.143
41–60	23 (65.7)	12 (34.3)	35	
61–80	16 (64.0)	9 (36.0)	25	
Total	42 (58.3)	30 (41.7)	72	

Analysis of radiologic patterns revealed that Usual Interstitial Pneumonia (UIP) was the most prevalent ILD subtype, present in 9 participants (12.5%), all of whom were ILD-positive (p < 0.001). Non-Specific Interstitial Pneumonia (NSIP) and Post-TB Fibrosis each accounted for 8.3% of cases, also confined to the ILD-positive group. Desquamative Interstitial Pneumonia (DIP) was found in 4 patients (5.6%), and Respiratory Bronchiolitis–ILD (RB-ILD) in 3 (4.2%), both exclusively among smokers with ILD. By contrast, small airway disease (8.3%), aspiration pneumonia, healed granulomatous infections, and pleural effusion patterns were predominantly observed in ILD-negative participants (p = 0.004). These results highlight the distinctive clustering of fibrotic HRCT findings among smokers, in contrast to predominantly non-fibrotic or inflammatory processes among non-smokers.

When evaluated across age strata (Table 4), the ILD proportion increased progressively from younger to older participants. Although the trend was not statistically significant (p = 0.143), it supported the hypothesis that chronicity and cumulative exposure duration may amplify risk. No significant gender difference was observed, but male predominance in both smoking prevalence and ILD cases suggests an overlapping risk influence.

Taken together, these data demonstrate a robust statistical and clinical association between smoking status and the occurrence of ILD on HRCT, with smokers exhibiting a nearly threefold higher probability of interstitial disease than non-smokers. The HRCT patterns most strongly correlated with ILD—particularly UIP, NSIP, and DIP—occurred exclusively among ILD-positive participants, reinforcing HRCT's diagnostic value in differentiating fibrotic from non-fibrotic pulmonary conditions. These findings validate the hypothesis that tobacco exposure is a critical determinant of ILD prevalence and pattern manifestation detectable through high-resolution imaging.

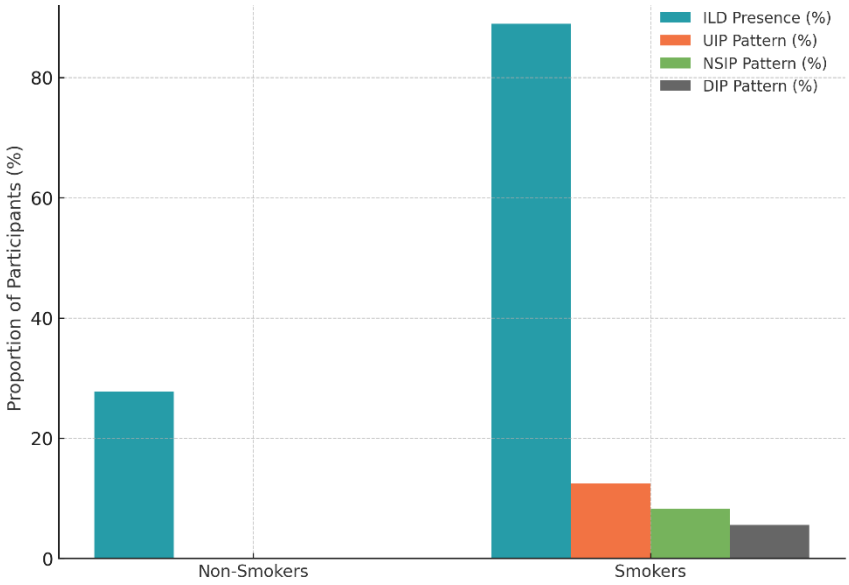

Figure 1 Distribution of ILD And Specific HRCT Patterns Among Smokers and Non-Smokers

The figure demonstrates a clear gradient in HRCT-detected interstitial abnormalities between smokers and non-smokers. ILD prevalence was markedly higher among smokers (88.9%) compared with non-smokers (27.8%), illustrating a near threefold elevation in disease probability. Moreover, fibrotic patterns such as Usual Interstitial Pneumonia (UIP), Non-Specific Interstitial Pneumonia (NSIP), and Desquamative Interstitial Pneumonia (DIP) appeared exclusively in smokers, with proportions of 12.5%, 8.3%, and 5.6%, respectively, while absent among non-smokers. This asymmetric distribution underscores the strong pathological linkage between chronic tobacco exposure and fibrotic remodeling of the lung parenchyma. Clinically, the visualization highlights HRCT's sensitivity in differentiating smoking-related ILD phenotypes, revealing a distinct clustering of fibrotic subtypes in smokers that may signify accelerated alveolar injury and impaired epithelial repair mechanisms contributing to progressive interstitial disease.

DISCUSSION

This study established a significant association between cigarette smoking and the presence of interstitial lung disease (ILD) as detected through high-resolution computed tomography (HRCT), demonstrating that smokers had a markedly higher probability of ILD than non-smokers. The data showed that 88.9% of smokers exhibited ILD compared with only 27.8% of non-smokers, corresponding to a large effect size and highly significant p-value ($p < 0.001$). These findings align with established literature indicating that smoking is a critical etiologic and exacerbating factor in multiple ILD subtypes, including usual interstitial pneumonia (UIP), desquamative interstitial pneumonia (DIP), and respiratory bronchiolitis–associated interstitial lung disease (RB-ILD) (10). Mechanistically, tobacco smoke induces repetitive epithelial injury, macrophage activation, oxidative stress, and dysregulated fibroblast proliferation, all of which contribute to fibrotic remodeling of the alveolar interstitium and progressive loss of lung compliance (11).

When integrated with prior research, the observed HRCT pattern distribution reinforces the pathogenic continuum between smoking and fibrotic parenchymal transformation. Margaritopoulos et al. (12) and Maher (13) similarly reported that smoking-related ILDs frequently manifest with overlapping UIP and RB-ILD features, confirming the radiologic–pathologic correlation noted in our study. The predominance of UIP among ILD-positive participants (12.5%) and its absence in non-smokers strengthens the assertion that smoking potentiates fibrosis within susceptible lung segments. Notably, while age showed a nonsignificant trend toward higher ILD prevalence among older participants, this likely reflects cumulative exposure duration rather than age-related susceptibility alone (14). Gender differences were also nonsignificant, which may indicate that biological sex has a smaller role in ILD development compared with environmental exposure intensity.

The results substantiate HRCT as a diagnostic cornerstone in differentiating smoking-induced parenchymal changes from other causes of diffuse lung disease. Consistent with Travis et al. and the American Thoracic Society guidelines, HRCT provided high diagnostic yield, enabling pattern-based classification of ILD without the need for invasive tissue sampling in most cases (15). The exclusive presence of UIP, NSIP, DIP, and RB-ILD patterns among smokers, and their absence among non-smokers, underscores HRCT's capability to detect subtle fibrotic transformations early in disease evolution. Furthermore, these findings have clinical implications for screening and surveillance in chronic smokers presenting with persistent cough or exertional dyspnea, even in the absence of spirometric impairment, as early fibrotic changes on HRCT may warrant intervention or cessation counseling (16).

Despite these strengths, several limitations must be acknowledged. The study's cross-sectional design precludes causal inference, and the modest sample size (n=72) limits generalizability across broader populations. Smoking exposure was self-reported, which may have introduced recall bias. Additionally, environmental and occupational exposures, though partially controlled for, may have contributed residual confounding. Radiologic

interpretation, although conducted by blinded dual reviewers, inherently carries a degree of subjectivity despite consensus review. Longitudinal follow-up with quantitative HRCT densitometry and spirometric correlation could enhance temporal resolution of disease progression.

Nevertheless, this study adds to existing evidence by quantifying the magnitude of association between smoking and HRCT-confirmed ILD within a South Asian population, where limited data exist on radiologic ILD phenotypes in smokers. Clinically, the findings emphasize the need for structured HRCT-based screening in high-risk populations and highlight the importance of smoking cessation as a primary preventive measure against fibrotic lung disease. Future research should incorporate multicenter longitudinal designs, larger cohorts, and biomarker integration (e.g., KL-6, SP-D) to delineate molecular pathways underlying smoking-associated fibrosis and to evaluate HRCT's predictive capacity for treatment response and disease progression (17).

CONCLUSION

This study demonstrated a strong and statistically significant association between cigarette smoking and the occurrence of interstitial lung disease (ILD) as identified through high-resolution computed tomography (HRCT). Smokers were nearly three times more likely to exhibit ILD compared with non-smokers, with predominant radiological patterns including usual interstitial pneumonia (UIP), non-specific interstitial pneumonia (NSIP), and desquamative interstitial pneumonia (DIP). These findings confirm that smoking not only increases the risk of ILD but also alters its radiologic phenotype toward fibrotic patterns. The study underscores the diagnostic value of HRCT in early detection and pattern differentiation of ILD, enabling timely intervention and smoking cessation strategies to mitigate disease progression. Clinically, the results advocate routine HRCT surveillance in high-risk smokers presenting with chronic respiratory symptoms, while emphasizing the importance of preventive policies targeting tobacco exposure to reduce ILD-related morbidity and mortality.

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