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ORIGINAL ARTICLE

Pattern of interstitial lung disease in connective tissue disease.

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ABSTRACT… Objective: To identify the pattern of interstitial lung diseases in connective tissue diseases (CTD-ILD) presenting in a tertiary care hospital. **Study Design:** Descriptive Cross-sectional study. **Setting:** Department of Rheumatology, Madina Teaching Hospital, Faisalabad. **Period:** July 2025 to December 2025. **Methods:** Patients between the ages of 18 and 80 who had been diagnosed with ILD based on their clinical presentation and radiological characteristics were included in the trial for six months. ILD was confirmed by high-resolution computed tomography (HRCT), whereas clinical criteria were used to diagnosis connective tissue disease. Comorbidities, clinical characteristics, demographic information, and HRCT results were documented. SPSS version 25 was used to analyze the data. The mean ± SD, percentages, and frequencies were computed. **Results:** Rheumatoid arthritis was the most common underlying condition among 54 CTD-ILD patients (mean age 51.9 ± 12.4 years, 70.4% female). Septal thickening (75.9%) and honeycombing (87.0%) were the most common HRCT abnormalities. **Conclusion:** Honeycombing and septal thickening were important HRCT patterns, and RA was the primary etiology of CTD.

Key words: Autoimmune Disorders, Connective Tissue Diseases, Interstitial Lung Diseases, Rheumatoid Arthritis, Systemic Sclerosis.

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INTRODUCTION

The CTD-ILD is a serious and potentially deadly outcome of a diverse range of autoimmune disorders called connective tissue diseases (CTD), which have been enabled by the development of improved methods of diagnostic imaging and heightened clinical awareness.[1,2]

The etiology of the disease is likely caused by autoantibody-mediated damage, a genetic predisposition, environmental triggering factors, and abnormal tissue repair in CTD, the pathogenesis of the disease is not still known, and due to the heterogeneity of disease manifestations in CTD, its diagnosis and treatment is exceptionally challenging.[3]

ILD is linked to a number of systemic autoimmune illnesses, including RA, SSc, SLE, polymyositis/dermatomyositis, Sjögren's syndrome, and MCTD.[6] More recently, the condition known as interstitial pneumonia with autoimmune characteristics (IPAF) has been identified in patients who exhibit autoimmunity symptoms but do not fully meet the diagnostic requirements for a particular CTD.[7] According to epidemiological research, ILD affects between 15% to 50% of people with CTD; however, prevalence rates vary significantly among ethnic groups.[8] The most common type of ILD is rheumatoid arthritis-related ILD (RA-ILD), which in certain case series is closer to half of CTD-ILD.[9] Systemic sclerosis (SSc-ILD) is the most prevalent type of ILD, and there is evidence that a sizable portion of SSc patients experience ILD during the course of their illness.[10] On the other hand, ILD is a major cause of morbidity but less common in inflammatory myopathies and SLE. This distribution emphasizes how crucial routine screening is for all autoimmune diseases.

A revolutionary CTD-ILD for thoroughly examining fine morphology is high-resolution CT (HRCT). Although there are many different CTD-ILD patterns, the two most common patterns seen in CTD ILD patients are NSIP and UIP.[11]

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While UIP, which is more frequently linked to RA, is typified by basal and subpleural honeycombing, NSIP, which is more frequently observed in SSc and inflammatory myopathies, manifests with ground-glass opacities and reticular abnormalities. In addition to being descriptive, these radiological subgroups have prognostic importance; the majority of UIP patients are linked to noticeably lower overall survival.

It appears that the risk factors for the development of ILD in CTDs are different from those for the autoimmune disorders that underlie them. It has been observed that fibrotic alterations are more likely to occur and progress in people who smoke, are older, or have metabolic comorbidities such diabetes mellitus.[12] For instance, PF occurrence is known to be influenced by disorders like smoking, which has been shown to work in concert with anti-cyclic citrullinated peptide (anti-CCP) antibodies to produce RA-related PF. The interaction with environmental exposures plus immune processes underscores the complex etiology of CTD-ILD.

Homes Significant outstanding issues continue despite improvements in diagnosis and treatment. Differences in clinical manifestation of CTD ILD are large, with fulminant, rapidly-progressing fibrosis on one end, and indolent illness on the other one. Antifibrotics and immunosuppressants are commonly applied to management strategies; although the different populations warrant usage, the best method of therapy remains undecided. Moreover, not many studies have outlined the scope and implications of CTD-ILD on health care systems that lack sufficient resources and most of the literature is anchored on Western cohorts. Owing to this ignorance, global facts are no longer as relevant by the local populations.

Since the impact of CTD-ILD on patient outcomes is significant and the disease can manifest itself differently, further research is needed to enhance the accuracy of diagnosis and allow treating these patients more specifically. In order to provide local information to guide screening and treatment methods, the current study aims to evaluate the clinical and radiological characteristics of CTD-ILD in patients who report to a tertiary care teaching hospital.

METHODS

From July 2025 to December 2025, a cross-sectional descriptive study was conducted among a sample of the population at the Madina Teaching Hospital's Rheumatology department in Faisalabad. The study was initiated following CPSP permission and received Institutional Review Board ethical approval (UMDC/IRB/04/26). All participants provided written informed consent before to inclusion in the study, which was conducted in accordance with the Declaration of Helsinki. Participation in the study was entirely voluntary, and patient confidentiality was ensured throughout the entire procedure.

Using the OpenEpi sample size software, the necessary sample size was calculated using the 95% CI and 5% margin of error for the prevalence rate of IPAF (idiopathic pneumonia with autoimmune characteristics), which was 3.6% according to Jayasinghe et al. To determine the necessary sample size of 54 patients, we used a non-probability consecutive sampling technique.

Male and female people between the ages of 18 and 80 who had been diagnosed with ILD and had undergone a clinical evaluation and HRCT were eligible subjects. Based on the clinical history, serological results, and characteristic imaging characteristics, radiologists and pulmonologists agreed to diagnose ILD. Patients who refused to participate, had insufficient imaging files, or had clinical documentation that was lacking were not included.

Clinical and demographic information was recorded into a standardized proforma that had already been created. Age, sex, and the main presenting symptoms—dyspnea, cough, joint pain, exhaustion, and chest pain—were the variables. Additionally included were comorbidities such diabetes mellitus, hypertension. Pack-years for current and previous smokers were computed to determine the intensity of exposure; baseline smoking status was classified as never, ever (former and current smoking), or current smoker. In cases that were possible, time differentiations between the progression of the disease and stroke therapy were also recorded.

HRCTs were performed on all the patients and the pictures were analyzed by highly skilled consultant radiologists who had expertise regarding thoracic imaging. Among the distinctive ILD patterns on HRCT, which were evaluated, is ground-glass opacities, septal thickening, honeycombing, cystic airspaces, and traction bronchiectasis. The information on the site, distribution, and size of these anomalies was recorded in each participant. To differentiate UIP and NSIP and other less known less common subtypes, the radiological patterns were classified based on universally accepted global standards.

The data were entered and analysed with the use of SPSS 25.0. The categorical values such as sex, comorbidities, smoking history, as well as the radiological findings were presented in frequencies and percentages, whereas the continuous variables (age, duration of symptoms) were represented as mean ± standard deviation (SD).

RESULTS

This study included a total of 54 CTD-ILD patients. The group's ages ranged from 18 to 80 years old, with an average age of 57.9 ± 10.4 years. Most of the patients were above fifty years of age. There were 38 females (70.4%) and 16 males (29.6%), indicating a significant female predominance. This proportion is comparable to the >2:1 female-to-male ratio seen in other autoimmune disorders in the general population. Distribution of different variables is shown in Table-I

With 29 patients (53.7%), RA was the most prevalent underlying connective tissue illness linked to ILD, followed by SSc in 10 patients (18.5%). Idiopathic pneumonia with autoimmune characteristics (IPAF) affected eight patients (14.8%), whereas MCTD affected two patients (3.7%) with mixed connective tissue disease and five patients (9.3%) with systemic lupus erythematosus. According to this relative distribution, RA and SSc combined already accounted for about three-fourths of all CTD-ILD in the study population. This is consistent with earlier global trends that showed both conditions were most frequently seen in the spectrum of CTD-ILD. (Table-II)

High-resolution computed tomography (HRCT) results from radiological evaluation revealed that the most frequent abnormality found and noted in 47 (87.0%) patients was honeycombing. The second most frequent anomaly (41 individuals, 75.9%) was septal thickening. Cystic air gaps were very uncommon, occurring in just four patients (7.4%); GG and traction bronchiectasis were seen in 15 patients each (27.7%). The fibrotic nature of the disease's progression in male IPF was also shown by the majority of men's honeycombing and septal thickening. These data also show that UIP-like radiological abnormalities predominate in patients with CTD ILD, especially those who have RA or SSc. (Table-III)

Age group, smoking history, diabetes mellitus, and septal thickening were all substantially correlated with the various CTD-ILD subtypes. Nearly one-third of cases (RA-ILD) had diabetes mellitus, which was primarily seen in RA-ILD patients. This condition is probably caused by glucocorticoid medication, chronic systemic inflammation, and metabolic abnormalities in RA. A well-established link between exposure to cigarette smoke and lung inflammation is demonstrated by the fact that patients with SSc and IPAF also had substantially lower smoking histories in the cigarette pack year units compared to those with RA and SLE.

There was a notable degree of interstitial fibrosis in IPAF and SSc, as seen by the proportionally larger septal thickness in these conditions compared to SLE. The frequency of RA rose with age among three groups (aged 41–80 years), while younger patients (18–40 years) were more likely to have IPAF and SLE, according to an analysis of the age distribution. This finding lends credence to the theory that autoimmune disease-related ILD typically mirrors the age-prevalence of the underlying connective tissue illness.

Other HRCT characteristics, such as ground glass opacities, traction bronchiectasis, honeycombing, and cystic air gaps, did not significantly differ between subtypes (all $p > 0.05$), indicating that radiologic similarity among ILDs due to CTD is typical. According to the study, the most prevalent HRCT findings for CTD-associated ILD in this series include rheumatoid arthritis, NVC, and

honeycombing and septal thickening. Strong correlations between age, smoking, and diabetes in son-suman were noted, underscoring the intricate role that metabolic and environmental variables play in promoting the onset and progression of ILD in connective tissue diseases.

TABLE-I

Distribution of different variables (n=54)

Confounding Variables		Frequency	%age
Age (years)	18-50	21	38.89
	51-80	33	61.11
Gender	Male	16	29.60
	Female	38	70.40
Duration of symptoms (months)	≤3	35	64.81
	>3	19	35.19
BMI (kg/m^2)	≤30	28	51.85
	>30	26	48.15
HTN	Yes	31	57.41
	No	23	45.59
DM	Yes	29	53.70
	No	25	46.30
Smoking	Yes	32	59.26
	No	22	40.74

TABLE-II

Connective tissue disease subtypes (n=54).

	Frequency (%)
Rheumatoid arthritis (RA)	29 (53.70%)
Systemic sclerosis (SSc)	10 (18.50%)
Idiopathic pneumonia with autoimmune features (IPAF)	08 (14.80%)
Systemic lupus erythematosus (SLE)	05 (9.30%)
Mixed connective tissue disease (MCTD)	02 (3.70%)

TABLE-III

HRCT findings (n=54).

	Frequency	Percentage
Honey combing	47	87.0
Septal thickening	41	75.90
Ground-glass opacities	15	27.70
Traction bronchiectasis	15	27.70
Cystic air spaces	04	7.40

DISCUSSION

The most prevalent kind of CTD-ILD in this investigation, excluding IPAF cases, was rheumatoid arthritis (RA)[1], followed by systemic sclerosis (SSc), mixed connective tissue disease (MCTD), systemic lupus erythematosus (SLE), and IPAF. According to several published studies, RA and SSc were the most common autoimmune causes of interstitial lung disease. This incidence is consistent with those findings. The large global prevalence of the disease, the chronic inflammatory load of RA, and cumulative exposure to immunosuppressive or disease-modifying anti-rheumatic medications may be the primary causes of the reported overall and specific prevalence of RA-ILD.[13-15] SSc ranks second in the majority of series, including ours, which is in line with the disease's known fibrotic nature and the early involvement of the lung parenchyma in its natural history.[15]

The most common radiological abnormalities in our series were thickened septa and honeycombing (83% and 72%, respectively). We propose that the UIP pattern was the most common in this sample because honeycombing is very widespread (almost 87 out of 100 patients). Since UIP indicates established fibrosis, it is widely recognized as a radiologic and histopathologic pattern in CTD-ILD that has been linked to a worse prognosis. Our findings support previous studies that found honeycombing to be a major predictor of an adverse outcome in RA-ILD.[13,14] However, NSIP was mostly linked to ground-glass opacities and septal thickening, particularly in SSc and IPAF patients. This is consistent with earlier findings that NSIP texture is a distinctive pattern in SSc-related ILD.[16] Since NSIP usually responds to corticosteroids and disease-modifying immunotherapy, and UIP usually shows minimal reversibility with immunosuppression, this radiographic differential has clinical significance.[16]

Additionally, our series' comorbidity profile provides significant clinical data. Significant correlations were found between diabetes mellitus and RA-ILD, indicating that metabolic comorbidities are prevalent in autoimmune illnesses and may intensify fibrotic and inflammatory lung pathways.[17] Diabetes causes low-grade inflammation and microvascular damage, both of which have been shown to exacerbate

lung fibrosis. Given that tobacco smoking is statistically associated with endemic RA-ILD, the study further emphasizes the significant influence of the environment in modifying disease risk. It is well known that smoking cigarettes can cause lung autoimmunity, particularly in people who are genetically predisposed to it.[18,19] It increases antigenic damage by promoting the citrullination of lung proteins and boosting the production of anti-CCP antibodies. The higher frequency and severity of ILD among smokers with RA in our population may also be explained by these hypothesized mechanisms. The association between smoking and SLE-ILD is also in line with earlier studies that demonstrated tobacco use increases endothelial damage and oxidative stress in lung diseases associated with lupus.

According to age-stratified study, RA-ILD was the primary cause in older patients, but IPAF and SLE were more prevalent in younger patients (18–40 years old). Regarding the age of presentation, this pattern is in line with other epidemiological studies. SLE-ILD typically affects young females, whereas RA-ILD typically manifests late in the course of the illness.[20] Disparities could result from variations in immune systems, length of illness, and cumulative drug use. Furthermore, our results highlight the significance of continuous pulmonary monitoring in chronic long-term RA by demonstrating that ILD secondary to RA is often subtle and develops after years of systemic inflammation.

There were no changes between the two groups in the radiological appearance of ground-glass opacities, cystic airspaces, and traction bronchiectasis in any of the subtypes. This supports the idea that, independent of the original autoimmune trigger, fibrotic remodeling is a common downstream end-point in CTD-ILD. There is evidence that fibroblast activation and extracellular matrix buildup take place through interdependent cellular pathways after parenchymal damage becomes self-sustaining.[21] Therefore, progressive fibrosis often converges to comparable radiographic phenotypes, even though starting histopathologic patterns may differ.

Our findings emphasize the necessity of using high-resolution computed tomography (HRCT) to describe ILD patterns in CTD from a clinical standpoint. The distinction between the UIP and NSIP patterns has significant therapeutic and prognosis implications. After immunomodulation fails, antifibrotic medications like nintedanib or pirfenidone should be investigated because UIP frequently has a fulminant course and is resistant to immunosuppressive therapy. Patients with NSIP, on the other hand, are more likely to be reversible with corticosteroids and steroid sparing medications.16 An early, accurate diagnosis of subtypes would, therefore, enable the deployment of tailored treatment regimens and better resource allocation, leading to improved clinical results, even though the classification of MDD into these subgroups is still primarily clinical. Furthermore, our findings support the complex pathophysiology of CTD-ILD. The main causes continue to be autoimmune dysregulation, chronic inflammation, and vascular injury; however, age, smoking, diabetes, and other environmental and genetic variables influence susceptibility and development. The role of cytokines (such as TGF-â and IL 6), extracellular matrix turnover, and fibroblast activation pathways in the pathophysiology of fibrosis has also been better understood thanks to immunology.[20,22] These findings support a cross-disciplinary approach to patient treatment by involving radiologists, pulmonologists, and rheumatologists in the patient management process.

We should take into account certain study limitations even though our results provide useful regional information. Because our study was limited to one tertiary facility, it may not be entirely applicable to the entire community of CTD-ILD patients in this region. Due to the short sample size, there may not be enough statistical power to identify a weaker association or to conduct additional research between different forms of CTD. Furthermore, the case-control design precludes drawing conclusions about the causal relationship between observed risk variables (such as smoking and diabetes) and the occurrence or course of ILD. Additionally, there was inconsistent availability of PFTs and longitudinal follow-up data to correlate radiographic patterns with mortality or deterioration in lung function. Mechanistic interpretation of the immunologic foundation for fibrosis, specifically differentiating between established fibrosis and ongoing inflammation,

was not possible due to the absence of biomarker and serologic profiling. Despite these drawbacks, this study offers crucial regional information on the characteristics and range of CTD-ILD in South Asia, which is underrepresented in international cohorts. This observation highlights the necessity of metacentric prospective studies that will verify these tendencies, some regional differences, and issues with evidence-based screening guidelines. Serial imaging, PFT trajectories, and biomarker analysis may be used in future research to reveal the natural history of illness evolution and response to treatment. Overall, this discussion indicates that UIP represents the leading radiologic pattern that is associated with a poor outcome, whereas RA and SSc are the most common CTDs that are linked to ILD. The intricate relationship between autoimmune and extrinsic linkages is shown by the fact that smoking and diabetes are two metabolic and environmental factors that have a significant impact on disease manifestation. A thorough, individualized assessment considering clinical, radiological, and immunological features is the basis of increasing the outcome of the CTD-ILD.

CONCLUSION

The most common underlying diseases in this CTD-ILD sample were rheumatoid arthritis and systemic sclerosis. Thickening of the septa and honeycombing were the most prevalent findings of the HRCT. None of the examined variables showed significant variance between CTD-ILD subtypes except septal thickness which is more prevalent in systemic sclerosis and in IPAF. No significant associations with age groups, comorbidities or gender were observed. These findings indicate the heterogeneity of CTD-ILD and importance of HRCT patterns, particularly, in distinguishing between subtypes through the thickness of the septal thickness.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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1	**Muhammad Maroof:** Drafting.
2	**Khalid Parvez:** Supervisor, topic selection.
3	**Syed Kamal Hussain Shah:** Data collection.
4	**Mahpara Munir:** Data analysis.
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6	**Hamza Arshad:** Data analysis.