Original Article **Open Access**

Characteristics of Connective Tissue Diseases Interstitial Lung Diseases (CTD-ILD) Presenting in a Tertiary Care Hospital

Feroz Raza, Faisal Faiyaz Zuberi, Arif ul Islam, Asim Shafeeque

Abstract

Objective: To determine the characteristics of connective tissue diseases–interstitial lung diseases (CTD-ILD) presenting in a tertiary care hospital.

Study design and setting: This descriptive cross-sectional study was conducted at the Department of Pulmonology, Ojha Institute of Chest Disease, Dow University of Health Sciences, Karachi, from 11th February 2025 till 26th July 2025.

Methodology: For 6 months study patients aged 18–80 years diagnosed with ILD on the basis of clinical presentation and radiological features were included. High-resolution computed tomography (HRCT) was performed to confirm ILD, while connective tissue disease diagnoses were based on clinical criteria. Demographic data, clinical features, comorbidities, and HRCT findings were recorded. Data were analyzed using SPSS version 25. Frequencies, percentages, and mean ± SD were calculated. Chi-square test was applied for categorical variables with p = 0.05 considered significant.

Results: Among 54 CTD-ILD patients (mean age 51.9 ± 12.4 years, 70.4% females), rheumatoid arthritis (53.7%) was the most common underlying disease. Honeycombing (87.0%) and septal thickening (75.9%) were the predominant HRCT abnormalities. On stratified analysis, diabetes mellitus (p = 0.026) and smoking (p = 0.021) were more frequent in RA-ILD. Younger patients (18–40 years) more often had IPAF and SLE compared to older patients (p = 0.002).

Conclusion: RA was the main cause of CTD-ILD, with honeycombing and septal thickening as key HRCT patterns. CTD-ILD subtypes were significantly linked to diabetes, smoking, septal thickening, and age.

Keywords: Autoimmune Disorders; Connective Tissue Diseases; Interstitial Lung Diseases; Rheumatoid Arthritis; Systemic Sclerosis.

How to cite this Article:
Raza F, Zuberi FF, Islam AU, Shafeeque A. Characteristics of Connective Tissue Diseases Interstitial Lung Diseases (CTD-ILD) Presenting in a Tertiary Care Hospital. J Bahria Uni Med Dental Coll. 2026;16(1):101-106 DOI: https://doi.org/10.51985/JBUMDC2025757

INTRODUCTION

Connective tissue diseases (CTDs) are a diverse group of autoimmune diseases characterized by systemic inflammation, immune dysfunction, and multi-organ damage.[1] An important and serious complication, interstitial lung disease (ILD) is responsible for a high level of morbidity and mortality.[2] The development of diagnostic imaging techniques and enhanced clinical awareness, have been responsible for delineating CTD-ILD as a major determinant for prognosis in patients with rheumatic diseases.[3]

CTD-ILD consists in a wide constellation of autoimmune disorders involving the lung parenchyma with both interstitial features and progressive fibrosis.[4] Although the exact pathogenesis of the disease remains unclear, it is thought that autoantibody-mediated injury, genetic predisposition, environmental triggers, and aberrant tissue repair processes are involved in the pathogenesis of the disease.[5] The variety of CTD-ILD makes the diagnosis and treatment quite challenging, since the clinical features, imaging features, and disease courses differ from one CTD to another.

Several systemic autoimmune diseases associate with ILD, such as RA, SSc, SLE, polymyositis/dermatomyositis, Sjögren's syndrome and MCTD.[6] The entity of interstitial pneumonia with autoimmune features (IPAF) has been more recently described in patients with signs of autoimmunity but did not satisfy the complete diagnostic criteria for a specific CTD.[7] Epidemiological studies indicate that ILD occurs in some 15–50% of CTD patients, but prevalence rates differ markedly between ethnic groups.[8]

Rheumatoid arthritis-related ILD (RA-ILD) is the most frequent type, representing a proportion closer to half of

Feroz Raza *(Corresponding Author)*
Post-graduate Trainee, Department of Pulmonology
Dow University of Health Sciences
Email: drsoomro110@gmail.com

Faisal Faiyaz Zuberi
Professor & Head, Department of Pulmonology
Dow University of Health Sciences
Email: ffzuberi@gmail.com

Arif ul Islam
Post-graduate Trainee, Department of Pulmonology
Dow University of Health Sciences
Email: arifkhan1656@gmail.com

Asim Shafeeque
Senior medical officer, Department of Pulmonology
Dow University of Health Sciences
Email: doc.asim39@gmail.com

Received: 09-10-2025
1st Revision:25-11-2025
Accepted: 08-01-2026
2nd Revision: 10-12-2025

CTD-ILD in some case series.[9] The most common second type of ILD is associated with systemic sclerosis (SSc-ILD) where there is evidence that a significant subset of SSc patients develop ILD within the disease course.[10] Conversely ILD is rarer in SLE and inflammatory myopathies but is a significant cause of morbidity. This distribution highlights the importance of systematic screening in any autoimmune disease.

High-resolution CT (HRCT) is a game changer CTD-ILD where fine morphology is investigated in detail. The patterns of CTD-ILD are diverse, and NSIP and UIP are the two most frequent patterns encountered in patients with CTD-ILD.[11] NSIP, more commonly seen in SSc and inflammatory myopathies, presents with ground-glass opacities and reticular changes, whereas UIP, which is more commonly associated with RA, is characterized by basal and sub pleural honeycombing. These radiological subtypes are not only descriptive, but also have prognostic significance, with most cases of UIP associated with significantly worse overall survival.

Risk factors for ILD development in CTDs seem to differ from those for the underlying autoimmune diseases alone. It has been noted that smoking, aging and metabolic comorbidities (e.g. diabetes mellitus) increase susceptibility and progression of fibrotic changes.[12] For placebo, diseases such as smoking, which has been found to act synergistically with anti-cyclic citrullinated peptide (anti-CCP) antibodies in generating RA-related PF, are known to contribute to PF occurrence. This crosstalk between environmental exposures and immunological mechanisms underscores the complex etiology of CTD-ILD.

Despite the progress in diagnosis and therapy, important open questions persist. The clinical presentation of CTD-ILD varies considerably, with indolent disease seen at one extreme and fulminant, rapidly progressive fibrosis observed at the other. Management approaches often employ the use of immunosuppressant or antifibrotics, however the ideal therapeutic strategy has not been established as a result of variable populations. In addition, the bulk of the literature is based on Western cohorts, and few studies have documented the spectrum and outcomes of CTD-ILD in resource-limited health care systems. This lack of understanding has limited the relevance of global data to local populations.

In view of the significant impact of CTD-ILD on patient prognosis and the heterogeneity of disease presentation, more studies are warranted to enhance the accuracy of diagnosis and facilitate the individualized management of such patients. The objective of the current study is to assess the clinical and radiological profile of CTD-ILD in patients presenting to a tertiary care teaching hospital, and to provide local evidence to inform strategies for screening and treatment.

METHODOLOGY

This cross-sectional descriptive study was carried out among sample of population in the Department of Pulmonology, Ojha Institute of Chest Diseases, Dow University of Health Sciences (DUHS), Karachi for a duration of 6 months from 11th February 2025 to 26th July 2025. The study was started after the approval of the CPSP and it obtained ethical approval from the Institutional Review Board of DUHS (IRB approval no: IRB-3812-DUHS-Approval-2025/44 Date approval: 10th February 2025). The present study was performed in compliance with the Declaration of Helsinki; written informed consent was given by all participants before inclusion. Involvement in the study was voluntary, and confidentiality of patient information was guaranteed at every point of the process.

The required sample size was determined using the OpenEpi sample size software based on prevalence rate of IPAF (idiopathic pneumonia with autoimmune features) as 3.6% according to Jayasinghe et al.95% CI, and 5% margin of error. Sample size calculation we employed a non-probability consecutive sampling method to obtain the required sample size of 54 patients.

Eligible participants were both male and female adults aged 18-80 years who underwent a clinical evaluation and HRCT and had received the diagnosis of ILD. The diagnosis of ILD was reached by agreement between radiologists and pulmonologists based on clinical history, serological findings, and typical imaging features. Patients declined participation, were incompletely documented clinically and had insufficient imaging files were excluded.

Demographic and clinical data was entered in a predesigned structured proforma. Variables consisted of age, sex, and primary presenting symptoms including dyspnea, cough, joint pain, fatigue and chest pain. Comorbidities such as diabetes mellitus, hypertension, chronic kidney disease, hypothyroidism HF and TIPE were also collected. Baseline smoking status was defined as never, ever (former and current smoking), or current smoker; pack-years were calculated for former and current smokers to estimate the intensity of exposure. Time lapse from illness onset and treatment previous history in stroke where it was possible, were also reported.

All the patients had HRCT of the chest and images were reviewed by highly experienced consultant radiologists specialized in thoracic imaging. Characteristic ILD patterns on HRCT were evaluated, including ground-glass opacities, septal thickening, honeycombing, cystic airspaces and traction bronchiectasis. The information on the localization, distribution and size of these anomalies was recorded for individual subjects. Radiological patterns were classified following accepted international criteria for separating UIP, NSIP, and other more rare subtypes.

Data entry and analysis were done using IBM SPSS Statistics

for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). Continuous values such as age and duration of symptoms were expressed as mean ± standard deviation (SD) while categorical values such as sex, comorbidities, smoking history, and radiological findings were reported in frequencies and proportions. Stratification was used to adjust potential effect modifiers (age, sex, comorbidity status and duration of symptoms) in the inferential analysis. The relation between categorical variables was analyzed by Chi-square test or Fisher's exact test where the expected frequency in a cell was <5. Independent sample t-tests or one-way analysis of variance (ANOVA) were used for continuous variables when applicable. We considered a p-value < 0.05 as statistically significant.

All analyses were examined for accuracy and double entry of data was used to provide quality control. Interpretation of the findings took into account potential confounders and the cross-sectional nature of the study. The results may extend the general understanding of clinical and radiologic features of connective tissue disease-related interstitial lung disease (CTD-ILD) and idiopathic pneumonia with autoimmune features in the local population.

RESULTS

A sum of 54 CTD-ILD patients were enrolled in this study. The average age of the group was 51.9 ±12.4 years and the ages varied between 28 -80 years. The majority of the patients were older than 40 years. A marked female predominance presented 38 females (70.4%) and 16 males (29.6%), similar to a female-to-male proportion of >2:1 as observed in other autoimmune diseases in the general population.

RA was the most common underlying connective tissue disease associated with ILD, 29 patients (53.7%), followed by SSc in 10 patients (18.5%). Eight (14.8%) patients had idiopathic pneumonia with autoimmune features (IPAF), while 5 patients (9.3%) with systemic lupus erythematosus and 2(3.7%) with mixed connective tissue disease had MCTD. This relative distribution indicated that RA and SSc together already accounted for almost three fourths of all CTD-ILD in the study population, similarly to previous worldwide trends where both these conditions were most often from CTD-ILD spectrum. (**Table 1**)

On radiological evaluation, the high-resolution computed tomography (HRCT) findings showed honeycombing was the commonest abnormality detected and recorded in 47 (87.0%) patients. Septal thickening was the second most common abnormality (41 patients, 75.9%). GG and traction bronchiectasis were identified in 15 patients each (27.7%); cystic air spaces were relatively rare, found only in four patients (7.4%). Most men also displayed honeycombing and septal thickening indicating the fibrotic character of disease evolution in male IPF. These results also indicate the dominance of UIP-like radiological findings in CTD-ILD patients, particularly those with RA or SSc. (**Table 2**)

Among the CTD-ILD subtypes analyzed by sex, RA was also the most common diagnosis among men and women (56.3% vs 52.6%, p = 0.84). Similarly, the other subtypes like SSc, IPAF and SLE had nearly equal proportions of males and females. This suggests that despite the fact both ILD and CTD have a preponderance for being female predominant among patient with these diseases, the distribution of ILD phenotypes is more balanced within men and women at least after developing the disease. The most common comorbidities were hypertension (22.2%) and DM (17.0%). By various subtypes of CTD-ILD, there were no comorbidities which demonstrated a statistically significant difference (all p > 0.05). Of interest, diabetes mellitus was more common in the RA-ILD group compared with other groups highlighting a relatively increased metabolic burden. Hypertension was highly prevalent among all subtypes that may have more to do with age-related comorbidity rather than disease-specific association. The HRCT findings are analyzed in other CTD-ILD subgroups as well. Most of each subtype had honeycombing and septal thickening (p > 0.05). Ground-glass opacities and traction bronchiectasis were slightly more common in IPAF and SSc, whereas ?ndings of cystic air spaces appeared only occasionally, typically in the setting of end-stage FB. Although not significant, these differences indicate unique mechanisms of lung injury between autoimmune subsets. (**Table 3**) Diabetes mellitus (p = 0.026), smoking history (p = 0.021, septal thickening (p = 0.008) and age group (p= 0.002) were significantly associated with different CTD-ILD subtypes. Diabetes Mellitus occurred in almost a third of cases (RA-ILD) and was mostly observed among those with RA-ILD, likely secondary to chronic systemic inflammation, glucocorticoid therapy and metabolic derangements in RA. Patients with SSc and IPAF also had significantly less smoking history in the cigarettes pack year units than those with RA and SLE, reflecting a well-documented connection between cigarette smoke exposure and inflammation of the lung.

Septal thickening was more disproportionately higher in IPAF, SSc than in SLE, indicating a significant degree of interstitial fibrosis in these diseases. Analysis of age distribution also revealed that younger patients (18–40 years) were more likely to exhibit IPAF and SLE, while the frequency of RA increased with age among 3 group (aged 41–80 years). This result supports the idea that ILD related to autoimmune diseases tend to reflect the age-prevalence of the underlying connective tissue disease.

Other HRCT features including honeycombing, ground-glass opacities, traction bronchiectasis, and cystic air spaces showed no significant difference among subtypes (all p > 0.05), suggesting that radiologic resemblance across CTD-related ILDs is common. The distribution of the findings is shown in **Figure 1.** The study demonstrates rheumatoid arthritis as the predominant cause of CTD-associated ILD in this series followed by NVC, honeycombing and septal

thickening being commonest HRCT findings. Strong relationships with diabetes among son-suman and smoking, and age were observed highlighting the complex role of metabolic and environmental factors in driving ILD development / progression in connective tissue diseases

DISCUSSION

In this study, exclusive of IPAF cases, RA (rheumatoid arthritis) was the most common type of CTD-ILD [1], being followed by SSc (systemic sclerosis), MCTD (mixed connective tissue disease), SLE (systemic lupus erythematosus); and then IPAF. This frequency is in agreement with a number of published series in which RA and SSc were reported as the most frequent autoimmune causes of interstitial lung disease. The observed overall and specific prevalence of RA-ILD may mainly be attributed to high global prevalence of disease, the contributing chronic inflammatory burden of RA, in combination with cumulative exposure to immunosuppressive or disease modifying anti-rheumatic therapies.[13,14,15] SSc occupies the second place in most series, such as ours, which is consistent with the well-established fibrotic nature of this disease and with the initial involvement lung parenchyma presents in its natural history.[15]

Radiologically, in our series honeycombing and thickened septa were the most frequent findings (83% and 72%). Since the UIP pattern, honeycombing is highly prevalent (almost 87 out of 100 patients) in this population we suggest that it was the most common pattern. UIP is commonly accepted as a radiologic and histopathologic pattern in CTD-ILD that has been associated with worse prognosis because it represents established fibrosis. Our results confirm at earlier research, reporting that honeycombing was a significant predictive factor for unfavorable outcome in RA-ILD.[13,14] On the other hand, septal thickening and ground-glass opacities were predominantly associated with NSIP, especially in SSc and IPAF patients and in accordance with previous reports of NSIP texture as a characteristic pattern in SSc-related ILD.[16] This radiographic distinction has therapeutic significance, as UIP typically represents low reversibility with immunosuppression while NSIP generally responds to corticosteroids and disease-modifying immunotherapy.[16]

The comorbidity profile of our series also offers relevant clinical information. Diabetes mellitus was substantially associated with RA-ILD, suggesting that metabolic comorbidities are common in autoimmune diseases and may amplify inflammatory and fibrotic lung pathways.[17] Diabetes leads to microvascular damage and low-grade inflammation,

Table 1. Distribution of CTD-ILD subtypes (n = 54)

Subtype	Frequency (n)	Percentage (%)
Rheumatoid arthritis (RA)	29	53.7
Systemic sclerosis (SSc)	10	18.5
idiopathic pneumonia with autoimmune features (IPAF)	8	14.8
Systemic lupus erythematosus (SLE)	5	9.3
Mixed connective tissue disease (MCTD)	2	3.7

Table 2. HRCT findings among participants (n = 54)

HRCT Feature	Frequency n (%)
Honeycombing	47 (87.0)
Septal thickening	41(75.9)
Ground-glass opacities	15(27.7)
Traction bronchiectasis	15(27.7)
Cystic air spaces	4(7.4)

Figure 1: HRCT features across CTD-ILD subtypes

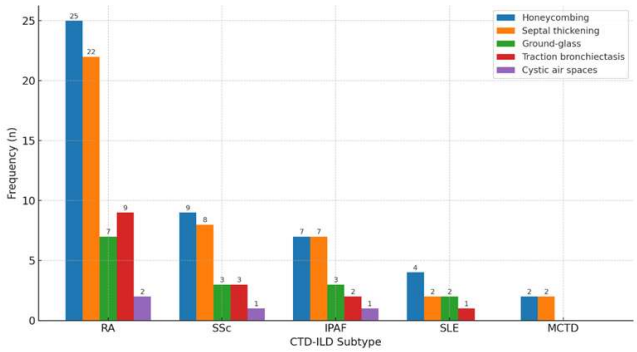

Table 3. Stratified analysis of variables with CTD-ILD subtypes (n = 54)

Variable	RA (n=29)	SSc (n=10)	IPAF (n=8)	SLE (n=5)	MCTD (n=2)	p-value
Gender						
Male (n=16)	9 (31.0%)	3 (30.0%)	2 (25.0%)	1 (20.0%)	1 (50.0%)	0.261
Female (n=38)	20 (69.0%)	7 (70.0%)	6 (75.0%)	4 (80.0%)	1 (50.0%)	
Comorbidities						
Hypertension (n=12)	6 (20.7%)	3 (30.0%)	2 (25.0%)	1 (20.0%)	0 (0%)	0.812
Diabetes mellitus (n=9)	8 (27.6%)	1 (10.0%)	0 (0%)	0 (0%)	0 (0%)	0.026
Hypothyroidism (n=5)	2 (6.9%)	1 (10.0%)	2 (25.0%)	0 (0%)	0 (0%)	0.054
CKD/Heart failure/PE (n=6)	3 (10.3%)	2 (20.0%)	0 (0%)	1 (20.0%)	0 (0%)	0.547
Smoking (n=8)	6 (20.7%)	1 (10.0%)	0 (0%)	1 (20.0%)	0 (0%)	0.021

both described to promote pulmonary fibrosis. The data also underscores the influential role of environment in modulating disease risk as the endemic RA-ILD is statistically linked to tobacco use. Cigarette smoking has been established as a potent trigger of pulmonary autoimmunity, especially in genetically susceptible individuals.[18,19] It drives citrullination of lung proteins and enhances the generation of anti-CCP antibodies, thereby enhancing antigenic damage. These putative mechanisms could also account for the more frequent and severe ILD in smokers with RA in our population. The relationship found between smoking and SLE-ILD is also consistent with previous research suggesting that tobacco exposure enhances oxidative stress and endothelial injury in lupus-related lung disease.

Age-stratified analysis demonstrated that IPAF and SLE were more common among younger patients (18–40 years), while RA-ILD was the main etiology in older patient. This pattern is consistent with previous epidemiological studies regarding the age of presentation in SLE-ILD, which affects usually young females, while RA-ILD tends to occur late in the course of the disease.[20] Differences may be due to differences in immune mechanisms, disease duration and cumulative medication use. In addition, our findings show that ILD secondary to RA is frequently insidious and occurs after many years of systemic inflammation, reinforcing the importance of ongoing pulmonary monitoring in chronic longstanding RA.

Radiologically, traction bronchiectasis and cystic airspaces, and ground-glass opacities were seen in all subtypes without differences between the two groups. This is compatible with the hypothesis that fibrotic remodeling is a downstream, common end-point in CTD-ILD, regardless of the initial autoimmune trigger. Evidence suggests that after parenchymal injury becomes self-sustained, fibroblast activation and extracellular matrix accumulation occur via interdependent cellular pathways.[21] And therefore, while initial histopathologic patterns could be different, advanced fibrosis frequently converges to similar radiographic phenotypes.

From a clinical perspective, our results highlight the need for high-resolution computed tomography (HRCT) to characterize ILD patterns in CTD. Differentiation of UIP and NSIP pattern has important prognostic and therapeutic relationship. UIP often follows a fulminant course and is refractory to immunosuppressive therapy; hence antifibrotic agents such as nintedanib or pirfenidone should be considered after immunomodulation failure. In contrast, patients with NSIP have more potential for reversibility with corticosteroids and steroid sparing agents.[16] Although the classification of MDD into these subgroups largely remains clinical, an early precise diagnosis of subtypes would, consequently, allow the application of individualized treatment strategies and better resource allocation leading to improved clinical outcomes.

In addition, our results also confirm the multifactorial pathogenesis of CTD-ILD. Autoimmune dysregulation, chronic inflammation and vascular injury remain primary drivers, but susceptibility and progression are modulated by genetic and environmental cofactors including smoking, diabetes and age. Immunology has also contributed to a better understanding of the involvement of fibroblast activation pathways, cytokines (e.g. TGF-â as well as IL-6) and extracellular matrix turnover in the pathophysiology of fibrosis.[20] Such observations favor a cross-disciplinary concept of care by including also rheumatologists, pulmonologists and radiologists into the patient management.

Although our results offer valuable regional information, we should consider some study limitations. Our study was conducted in a single tertiary center and might not fully apply to the general CTD-ILD patient population within this geographical area. The sample size was small and might preclude some statistical power to find weaker relationship or further analysis between distinct types of CTD. Additionally, the case-control design does not allow causal inferences for observed risk factors (like diabetes and smoking) on ILD incidence or progression. Furthermore, PFTs and longitudinal follow-up data were not consistently available for correlation of radiographic pattern with lung function decline or mortality. The lack of biomarker and serologic profiling precluded mechanistic interpretation of the immunologic basis for fibrosis, in particular distinguishing between active inflammation and established fibrosis. Notwithstanding these limitations, this work provides pivotal regional data on the spectrum and features of CTD-ILD in South Asia which is poorly represented in global cohorts. This observation highlights the necessity of metacentric prospective studies to validate these patterns, determine regional variability, and generate evidence-based screening guidelines. Subsequent studies using serial imaging, PFT trajectories and biomarker profiling will provide insights into the natural history of disease evolution and response to treatment. In conclusion, this study demonstrates that RA and SSc are still the most common CTDs associated with ILD, where UIP is the principal radiologic pattern related to poor outcomes. Diabetes and smoking are some of the metabolic and environmental agents that seriously affect disease expression, emphasizing the complex contribution of autoimmunity to extrinsic links. Holistic, personalized assessment incorporating clinical, radiological and immunological features is the backbone of enhancing outcome in CTD-ILD.

CONCLUSION:

In this cohort of CTD-ILD, rheumatoid arthritis was the most common underlying disease, followed by systemic sclerosis. Honeycombing and septal thickening were the predominant HRCT findings. Among all variables analyzed, only septal thickening showed a significant variation across CTD-ILD subtypes, being more frequent in systemic sclerosis

and IPAF. No significant associations were observed with gender, comorbidities, or age groups. These findings highlight the heterogeneity of CTD-ILD and emphasize the role of HRCT patterns, particularly septal thickening, in distinguishing subtypes.

Conflicts of interest: Nil

Source of Funding: Nil

Acknowledgement: Nil

Authors Contribution:
Feroz Raza: Study conception, design, and overall supervision.
Faisal Faiyaz Zuberi: Critical review of the manuscript and final approval for submission.
Arif ul Islam: Technical input and manuscript review.
Asim Shafeeque: Data verification, analysis support, and critical revision of the manuscript.

REFERENCES:

1. Mogyoróssy S, Nagy-Vincze M, Griger Z, Dankó K, Szabó Na, Szekanecz Z, Et Al. Novel Aspects Of Muscle Involvement In Immune-Mediated Inflammatory Arthropathies And Connective Tissue Diseases. Autoimmune Rev. 2023;22(5):103311. Doi: Https://Doi.Org/10.1016/ J.Autrev. 2023.103311.
2. Shi Y, Jiang N, Li M, Zeng X, Tian X. Mesenchymal Stem Cells And Connective Tissue Diseases: From Bench To Bedside. J Transl Int Med. 2022;11(1):30–45. Doi: Https://Doi.Org/10.1515/Jtim-2022-0004.
3. Chan C, Ryerson Cj, Dunne Jv, Wilcox Pg. Demographic And Clinical Predictors Of Progression And Mortality In Connective Tissue Disease-Associated Interstitial Lung Disease: A Retrospective Cohort Study. Bmc Pulm Med. 2019;19(1):192. Doi: Https://Doi.Org/10.1186/S12890-019-0955-Y.
4. Shao T, Shi X, Yang S, Zhang W, Li X, Shu J, Et Al. Interstitial Lung Disease In Connective Tissue Disease: A Common Lesion With Heterogeneous Mechanisms And Treatment Considerations. Front Immunol. 2021;12:684699. Doi: Https://Doi.Org/10.3389/Fimmu.2021.684699.
5. Matson Sm, Demoruelle Mk. Connective Tissue Disease-Associated Interstitial Lung Disease. Immunol Allergy Clin North Am. 2023;43(2):229–44. Doi: Https://Doi. Org/10.1016/ J.Iac.2023.01.003.
6. Cerro Chiang G, Parimon T. Understanding Interstitial Lung Diseases Associated With Connective Tissue Disease (Ctd-Ild): Genetics, Cellular Pathophysiology, And Biologic Drivers. Int J Mol Sci. 2023;24(3):2405. Doi: Https://Doi.Org/ 10.3390/Ijms24032405.
7. Yoo H, Hino T, Han J, Franks Tj, Im Y, Hatabu H, Et Al. Connective Tissue Disease-Related Interstitial Lung Disease (Ctd-Ild) And Interstitial Lung Abnormality (Ila): Evolving Concept Of Ct Findings, Pathology And Management. Eur J Radiol Open. 2020;8:100311. Doi: Https://Doi.Org/ 10.1016/ J.Ejro.2020.100311.
8. Atzeni F, Gerardi Mc, Barilaro G, Masala If, Benucci M, Sarzi-Puttini P. Interstitial Lung Disease In Systemic Autoimmune Rheumatic Diseases: A Comprehensive Review. Expert Rev Clin Immunol. 2018;14(1):69–82. Doi: Https://Doi.Org/10.1080/1744666x.2018.1411197.
9. Mira-Avendaño I, Rivera F, Rojas C, Gómez D, Martínez P. Interstitial Lung Disease In Systemic Sclerosis, Ra, Iim, Sle And Mctd: Relative Frequencies Of Ct Imaging Patterns. Rev Colomb Reumatol. 2024;31(1):45–53. Doi: Https://Doi.Org/ 10.1016/J.Rcreu.2023.07.002.
10. Jepsen Y, Alarcon M, Klein U, Thomsen A. Practical Guidance For Early Recognition And Follow-Up Of Patients With Ctd-Ild. Clin Rheumatol. 2024;43(2):317–25. Doi: Https://Doi.Org/10.1007/S10067-023-06584-Y.
11. Raghu G, Remy-Jardin M, Myers Jl, Richeldi L, Ryerson Cj, Lederer Dj, Et Al. Diagnosis Of Idiopathic Pulmonary Fibrosis: Ats/Ers/Jrs/Alat Guideline. Am J Respir Crit Care Med. 2018;198(5):E44–68. Doi: Https://Doi.Org/ 10.1164/ Rccm.201807-1255st.
12. Jayasinghe P, Wickramasinghe N, Krishnakumar P, Sadikeen A, Fernando A. Characteristics Of Connective Tissue Diseases Associated Interstitial Lung Diseases – A Single Centre Study In Sri Lanka. Ceylon Med J. 2021;66(1):38–43. Doi: Https://Doi.Org/10.4038/Cmj.V66i1.9405.
13. Antoniou Km, Margaritopoulos Ga, Goh Ns, Hansell Dm, Nicholson Ag. Honeycombing In Usual Interstitial Pneumonia: Prognostic Significance. Eur Respir J. 2016;48(5):1518–28. Doi: Https://Doi.Org/10.1183/13993003.01082-2016.
14. Kim Ej, Elicker Bm, Maldonado F, Webb Wr, Ryu Jh, Van Uden Jh, Et Al. Usual Interstitial Pneumonia In Rheumatoid Arthritis-Associated Ild. Arthritis Res Ther. 2010;12(2):R79. Doi: Https://Doi.Org/10.1186/Ar2987.
15. Yoo H, Hino T, Han J, Franks Tj, Im Y, Hatabu H, Et Al. Connective Tissue Disease-Related Interstitial Lung Disease And Interstitial Lung Abnormality: Evolving Concepts Of Ct Findings, Pathology, And Management. Eur J Radiol Open. 2020;8:100311. Doi: Https://Doi.Org/10.1016/ J.Ejro.2020 .100311.
16. Wells Au, Denton Cp. Interstitial Lung Disease In Systemic Sclerosis: Nsip Vs Uip. Rheumatology (Oxford). 2014;53(9): 1612–21. Doi: Https://Doi.Org/10.1093/ Rheumatology/ Keu165.
17. Kelly Ca, Saravanan V, Nisar M, Arthanari S, Woodhead Fa, Price-Forbes An, Et Al. Rheumatoid Arthritis-Related Interstitial Lung Disease: Associations With Diabetes And Other Comorbidities. Ann Rheum Dis. 2014;73(8):1511–9. Doi: Https://Doi.Org/10.1136/Annrheumdis-2012-203160.
18. Sparks Ja, Chang Sc, Liao Kp, Costenbader Kh, Karlson Ew. Associations Of Smoking And Rheumatoid Arthritis-Related Interstitial Lung Disease Risk. Arthritis Care Res (Hoboken). 2019;71(2):187–95. Doi: Https://Doi.Org/10.1002/Acr.23782.
19. Iqbal K, Kelly C. Interaction Of Smoking And Anti-Ccp In Rheumatoid Arthritis-Associated Ild. Rheumatology (Oxford). 2021;60(1):104–12. Doi: Https://Doi.Org/10.1093/ Rheumatology/Keaa223.
20. Singh N, Varghese J, Verma S, Agarwal V, Misra R. Interstitial Lung Disease In Systemic Lupus Erythematosus: Clinical Spectrum And Prognosis. Lupus. 2016;25(9):960–8. Doi: Https://Doi.Org/10.1177/0961203315622824.
21. Assayag D, Kim Ej, Elicker Bm, Collard Hr, King Te Jr. Survival Trends In Connective Tissue Disease-Related Interstitial Lung Disease: Influence Of Hrct Pattern. Chest. 2014;146(3):633–40.