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Correlation of Clinical, Hematological, Radiological and Spirometric Profile among Interstitial Lung Disease Patients Presenting To Tertiary Care Centre

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Abstract: Clinical, radiographic, and pulmonary function test (PFT) data in tertiary care patients with interstitial lung disease (ILD) will be compared and correlated. ILD is a heterogeneous collection of parenchymal lung illnesses with inflammation and fibrosis. In ILD patients, appropriate diagnosis, prognosis, and therapy planning need understanding the link between clinical symptoms, radiographic findings, and pulmonary function measurements. A retrospective study was performed on ILD patients' medical records who attended the tertiary care centre between [start date] and [end date]. Demographic, symptom, and comorbidity data were obtained. High-resolution computed tomography (HRCT) images were evaluated to determine lung involvement and fibrosis. Pulmonary function was assessed using PFT findings, including spirometry, lung volumes, and diffusion capacity. ILD patients have a variety of clinical manifestations, radiographic patterns, and pulmonary function abnormalities, according to preliminary results. Correlation analysis will reveal possible correlations between these measures, improving ILD diagnosis and treatment algorithms. Further study is needed to confirm these results and identify other ILD disease progression and treatment response variables.

Keywords: Interstitial lung disease (ILD), Clinical presentation, Radiological patterns, Pulmonary function tests (PFTs), Tertiary care center, High-resolution computed tomography (HRCT)

1. Introduction

Interstitial lung disease (ILD) is a heterogeneous set of parenchymal lung illnesses that cause lung interstitium inflammation and fibrosis, causing lung damage and gas exchange problems [1]. Due to varied clinical presentations, unpredictable illness trajectories, and limited treatment choices, many disorders are difficult to diagnose and treat. To accurately diagnose, prognose, and treat ILD patients, one must understand the complex relationship between clinical characteristics, radiological findings, and pulmonary function abnormalities. As we learn more about disease pathophysiology, ILD categorization becomes more complicated. ILDs have been classified by aetiology, histopathology, or clinical-radiology. Idiopathic pulmonary fibrosis (IPF) is a form of ILD that has increasing lung parenchyma fibrosis and a typical interstitial pneumonia (UIP) pattern on HRCT and histology [2]. Others, such connective tissue disease-associated ILD (CTD-ILD) and hypersensitivity pneumonitis (HP), have different clinical and radiological presentations, confounding diagnostic algorithms. Figure 1 elucidates the diverse manifestations encompassing fibrosis, scarring, and respiratory abnormalities within the lung parenchyma.

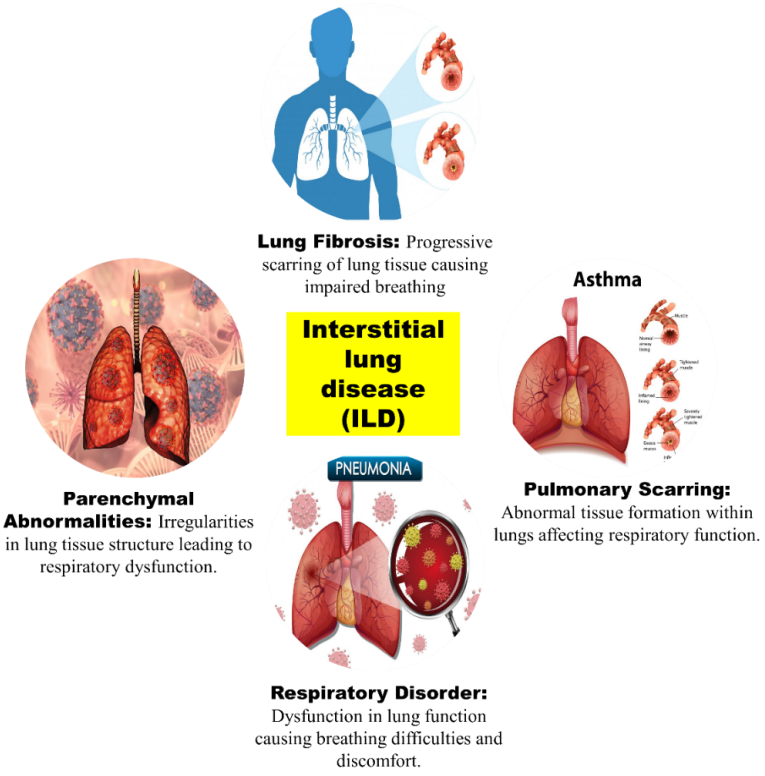

Figure 1. Key Characteristics of Interstitial Lung Disease: A Schematic Illustration

ILD may cause gradual dyspnea and non-productive cough or abrupt exacerbations with respiratory failure. Patients commonly have growing exertional dyspnea, which may lower quality of life and function. Extrapulmonary symptoms including digital clubbing, skin rash, and joint discomfort may indicate a connective tissue problem or environmental exposure [3]. ILD must be diagnosed thoroughly to separate it from other respiratory disorders like chronic obstructive pulmonary disease (COPD) or congestive heart failure. Radiological examination, especially HRCT, is crucial to ILD diagnosis and categorization. HRCT may examine lung parenchymal anomalies such ground-glass, reticular, and honeycombing, which are indicative of distinct ILD subtypes. Basal and subpleural reticulation with honeycombing on HRCT strongly suggests IPF. Similarly, radiographic patterns like non-specific interstitial pneumonia (NSIP) and organising pneumonia (OP) can subclassify ILDs and guide therapy [4]. Pulmonary function testing helps ILD patients estimate disease severity and track disease progression. ILD patients generally have a restrictive ventilatory dysfunction, with lower FVC and a preserved FEV1/FVC ratio, according to spirometry. Lung volumes, such as TLC and RV, also reveal lung constraint. In ILD patients, alveolar injury and fibrosis reduce diffusion capacity for carbon monoxide (DLCO), which measures alveolar-capillary membrane function. ILD is complicated and diverse, thus pulmonologists, radiologists, and pathologists must work together to diagnose and treat it. This study compares and correlates clinical, radiological, and pulmonary function tests in ILD patients presenting at a tertiary care centre to assess their diagnostic and prognostic value. We want to improve ILD pathogenesis and patient treatment by identifying parameter connections.

1.1 Background of Interstitial Lung Disease (ILD)

Interstitial lung disease (ILD) is a diverse collection of diffuse parenchymal lung illnesses that cause lung interstitium inflammation and fibrosis. This complicated entity includes several illnesses with different aetiologies, clinical manifestations, and prognoses, making diagnosis and treatment difficult. Accurate diagnosis, therapy, and patient outcomes depend on understanding ILD pathogenesis and clinical presentations [5]. The name "interstitial lung disease" comes from histological observations of inflammatory and fibrotic alterations in the lung interstitium, which comprises the alveolar epithelium, basement membrane, and perivascular and perilymphatic tissues. These changes disturb the fragile lung parenchyma, limiting gas exchange and causing respiratory insufficiency. ILD is characterised by interstitial inflammation, fibrosis, and architectural distortion, which may be categorised into histopathological patterns based on lung biopsy findings.

ILD incidence and prevalence vary internationally, with estimates ranging from 10 to 40 cases per 100,000 population per year. Sarcoidosis may afflict people of various ages and ethnicities, although idiopathic pulmonary fibrosis (IPF) is more common in older folks [6]. Environmental, occupational, and genetic variables have been linked to ILD, demonstrating its complex character. Our knowledge of disease pathophysiology and the creation of standardised diagnostic criteria have changed ILD categorization. ILDs were formerly classified by clinical-radiological patterns on imaging techniques such HRCT or lung biopsy histopathology. However, molecular and genetic indicators have changed ILD categorization to focus on underlying aetiologies and disease processes. The progressive and irreversible form of fibrosing interstitial pneumonia known as idiopathic pulmonary fibrosis (IPF) is one of the best researched ILD subtypes. On lung biopsy, IPF has patchy fibrosis with honeycombing and peripheral subpleural fibrosis, similar to typical interstitial pneumonia (UIP) [7]. IPF causes respiratory symptoms to deteriorate and ultimately to respiratory failure and mortality within 2 to 5 years after diagnosis. Besides IPF, additional ILD subtypes include CTD-ILD, HP, sarcoidosis, and NSIP. Each subtype has unique clinical, radiological, and pathological characteristics, requiring individualised diagnosis and therapy. CTD-ILD individuals may have underlying autoimmune disorders such rheumatoid arthritis or systemic sclerosis that need targeted immunosuppressive medication in addition to ILD care.

1.2 Significance of Comparative Analysis in Interstitial Lung Disease (ILD)

Comparing clinical, radiological, and pulmonary function tests (PFTs) in interstitial lung disease (ILD) patients helps us understand disease pathophysiology, improve diagnostic accuracy, guide treatment decisions, and improve patient outcomes. Clinicians may understand ILD and customise treatment by thoroughly comparing and correlating factors across diagnostic modalities [8]. Comparative analysis in ILD helps explain illness causation and development by revealing disease parameter connections. Clinical symptoms like dyspnea and cough may be linked to radiological abnormalities like ground-glass opacities or reticular patterns on high-resolution computed tomography (HRCT) to determine ILD subtypes and severity. Integrating spirometry, lung volumes, and diffusion capacity findings provides for a complete evaluation of lung function impairment and patient prognosis. Comparative study helps refine diagnostic algorithms and improve ILD diagnosis accuracy. Clinicians may identify diagnostic difficulties and possible confounders that may delay or misdiagnose ILD by assessing clinical, radiographic, and pulmonary function measures. A sophisticated strategy that incorporates the complete clinical and diagnostic range is needed to identify ILD from other respiratory disorders like COPD or congestive heart failure [9].

Comparative study also helps identify prognostic indicators and risk factors for ILD development and bad outcomes. Comparing baseline pulmonary function test results with longitudinal follow-up data may predict disease development and identify individuals at risk of acute exacerbations. Integrating radiographic indicators like HRCT fibrosis or traction bronchiectasis with clinical and functional characteristics might improve prognostic models and therapy classification. Comparative analysis aids ILD treatment choices and actions in addition to diagnostic and prognostic purposes. Clinicians may customise pharmacological and non-pharmacological therapies to patient features and illness phenotypes by linking disease parameters with therapy response and clinical outcomes [10]. Identifying ILD patients with growing fibrosis on HRCT and worsening pulmonary function may induce early commencement of antifibrotic medication like pirfenidone or nintedanib to slow disease progression and improve outcomes. Comparative analysis also encourages pulmonologists, radiologists, pathologists, and other ILD specialists to collaborate and share information. Multidisciplinary ILD care optimises resource utilisation, reduces diagnostic ambiguity, and enhances patient satisfaction by integrating knowledge from diverse disciplines and using complementary diagnostic techniques.

2. Interstitial Lung Disease: Overview

Interstitial lung disease (ILD) is a diverse collection of parenchymal lung illnesses that cause lung interstitium inflammation and fibrosis. This varied group includes several disorders with different aetiologies, clinical presentations, radiological patterns, and prognoses. Accurate diagnosis, proper care, and patient outcomes depend on understanding ILD's underlying concepts and traits. The name "interstitial lung disease" comes from histological studies of inflammatory and fibrotic alterations in the lung interstitium, which comprises the alveolar epithelium, basement membrane, and perivascular and perilymphatic tissues [11]. These changes disturb the fragile lung parenchyma, limiting gas exchange and causing respiratory insufficiency. ILD is characterised by interstitial inflammation, fibrosis, and architectural distortion, which cause clinical symptoms and functional deficits. ILDs are categorised by clinical, radiological, and histological criteria, with aetiology a major factor. IPF is a well-known ILD subtype that causes progressive and irreversible lung parenchyma fibrosis with a typical interstitial pneumonia (UIP) pattern on high-resolution computed tomography (HRCT) and histology. CTD-ILD, HP, sarcoidosis, and NSIP are further ILDs with different clinical and pathological characteristics. Table 1

categorizes and describes various subtypes of Interstitial Lung Disease (ILD), outlining their characteristics and typical presentations to aid in diagnostic differentiation and clinical management.

Table 1. Categorization and Interpretation of Subtypes of Interstitial Lung Disease (ILD)

ILD Category	Description	Examples
Idiopathic Pulmonary Fibrosis (IPF)	Chronic, progressive fibrosing interstitial pneumonia of unknown cause, predominantly affecting older adults.	Usual Interstitial Pneumonia (UIP), Honeycombing
Nonspecific Interstitial Pneumonia (NSIP)	Uniform interstitial inflammation and fibrosis with less heterogeneity than UIP, often with better prognosis.	Cellular and Fibrotic Patterns
Cryptogenic Organizing Pneumonia (COP)	Subacute pneumonia-like illness characterized by granulation tissue plugs within alveolar ducts and spaces.	Organizing Pneumonia Pattern, Patchy Infiltrates
Acute Interstitial Pneumonia (AIP)	Rapid-onset interstitial lung disease with severe hypoxemia and diffuse alveolar damage on histopathology.	Diffuse Alveolar Damage (DAD), Hyaline Membrane
Connective Tissue Disease-ILD (CTD-ILD)	ILD occurring in conjunction with underlying autoimmune conditions such as rheumatoid arthritis or scleroderma.	Rheumatoid Lung, Scleroderma Interstitial Lung Disease (ILD)
Hypersensitivity Pneumonitis (HP)	Immunologically mediated lung disease triggered by inhalation of various organic antigens or particles.	Farmer's Lung, Bird Fancier's Lung
Occupational Lung Diseases	ILD resulting from exposure to occupational hazards such as asbestos, silica, or coal dust.	Asbestosis, Silicosis, Coal Worker's Pneumoconiosis
Drug-Induced Interstitial Lung Disease	ILD caused by adverse reactions to medications, often presenting with diffuse alveolar damage or hypersensitivity reactions.	Chemotherapy-Induced ILD, Amiodarone Lung Toxicity
Idiopathic Interstitial Pneumonias (IIPs)	A group of ILDs characterized by diffuse alveolar damage and fibrosis without a known cause or association.	Acute Fibrinous and Organizing Pneumonia (AFOP), Desquamative Interstitial Pneumonia (DIP)

ILD symptoms vary and may include exertional dyspnea, non-productive cough, lethargy, and exercise intolerance. Symptoms might start slowly and worsen to severe exacerbations with fast respiratory function loss. Extrapulmonary symptoms including digital clubbing, skin rash, and joint discomfort may indicate a systemic illness or environmental exposure. A thorough diagnostic strategy is needed to separate ILD from other respiratory disorders with comparable clinical manifestations. HRCT is the preferred imaging modality for diagnosing and classifying ILD [12]. HRCT may examine lung parenchymal anomalies such ground-glass, reticular, and honeycombing, which are indicative of distinct ILD subtypes. HRCT with basal and subpleural reticulation and honeycombing strongly suggests IPF. Radiological features including NSIP and organising pneumonia (OP) help subclassify ILDs and guide therapy. Pulmonary function testing helps ILD patients track illness and therapy response by objectively measuring lung function deterioration. ILD patients generally have a restrictive ventilatory dysfunction, with lower FVC and a preserved forced expiratory volume in one second (FEV1) and forced vital capacity (FVC), according to spirometry. Lung volumes and DLCO diffusion capacity help explain ILD's lung limitation and gas exchange problems.

2.1 Definition and Classification of Interstitial Lung Disease (ILD)

Interstitial lung disease (ILD) is a collection of parenchymal lung illnesses that include inflammation and fibrosis of the lung interstitium, which includes the alveolar epithelium, basement membrane, and perivascular and perilymphatic tissues. These pathological alterations disturb the delicate lung parenchyma architecture, limiting gas exchange and causing respiratory insufficiency. Etiological, clinical, radiological, and histological variables are used to classify ILD. Idiopathic ILDs are a subgroup of illnesses with unclear causes in one way to classify ILDs. IPF, for instance, is characterised by increasing lung parenchyma fibrosis without a reason [13]. In addition to NSIP, cryptogenic organising pneumonia is another idiopathic ILD.

Histopathological features on lung biopsies may help classify ILDs by disease entity. IPF is linked to UIP pattern, which includes patchy fibrosis with honeycombing and peripheral subpleural fibrosis. In contrast, NSIP and some connective tissue disease-associated ILDs have homogeneous interstitial inflammation and fibrosis. Clinical-radiological categorization of ILD requires comparing clinical characteristics to HRCT results. Ground-glass, reticular, and honeycombing radiological patterns indicate ILD subtypes. OP may show patchy consolidations with a peripheral distribution, whereas NSIP has bilateral ground-glass and reticular opacities on HRCT. ILDs may also be categorised by systemic disorders or environmental exposures. CTD-ILDs are ILDs that arise in autoimmune illnesses such rheumatoid arthritis or systemic sclerosis [14]. Organic dusts, moulds, and aerosolized antigens may cause hypersensitivity pneumonitis (HP), another ILD.

2.2 Pathophysiology of Interstitial Lung Disease (ILD)

Interstitial lung disease (ILD) is caused by a complicated chain of inflammatory and fibrotic processes that damage the lung parenchyma and hinder gas exchange. ILD development depends on subtype and aetiology, however some common pathways contribute to disease pathogenesis. Dysregulated immune response, which activates inflammatory cells and releases pro-inflammatory cytokines and chemokines in the lung interstitium, is fundamental to ILD pathogenesis. Inflammatory cells such macrophages, lymphocytes, and neutrophils invade the lung parenchyma after environmental or autoimmune assaults. Immune cells release cytokines such tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and transforming growth factor-beta (TGF-β), leading to inflammation and tissue damage [15].

Collagen, fibronectin, and elastin accumulate excessively in the lung interstitium due to inflammation. Profibrotic signals cause fibroblastic foci and collagen-rich scar tissue because fibroblasts, myofibroblasts, and activated epithelium cells proliferate and alter phenotype. Fibrosis alters lung architecture, alveolar gas exchange, and function. Epithelial damage and dysfunction are also increasingly linked to ILD aetiology. Epithelial cells on the alveolar surface protect the lung tissue from the outside world. Environmental assaults, oxidative stress, or autoimmune injury to the alveolar epithelium may cause abnormal healing mechanisms and fibrosis. Dysregulated epithelial-mesenchymal interaction, including abnormal activation of Wnt/β-catenin and TGF-β pathways, promotes fibrotic responses and disease development.

Epigenetic changes and genetic vulnerability also contribute to ILD development. Several genetic variations enhance ILD risk or severity and development. Mutations in genes producing surfactant proteins (like SFTPC, SFTPA2) or telomere-related genes (like TERT, TERC) have been linked to familial ILD and sporadic IPF. Epigenetic changes such DNA methylation, histone acetylation, and microRNA dysregulation may alter gene expression and cause immunological and fibrotic dysregulation in ILD [16]. ILD pathogenesis also involves occupational risks, air contaminants, and infectious pathogens. Silica, asbestos, and organic dusts may cause lung parenchyma inflammation and fibrosis, causing pneumoconiosis or hypersensitivity pneumonitis. Viral infections like Epstein-Barr virus (EBV) or herpesvirus may also cause ILD, especially in those with immunological dysregulation.

2.3 Epidemiology of Interstitial Lung Disease (ILD)

Understanding interstitial lung disease (ILD) epidemiology helps identify at-risk individuals, understand disease trends, and drive preventative and care measures. ILD is a broad collection of parenchymal lung illnesses that cause lung interstitium inflammation and fibrosis. Its causes, symptoms, and prognoses vary. Epidemiological research has illuminated the worldwide health burden of ILD by revealing its incidence, prevalence, risk factors, and consequences [17]. ILD epidemiology varies by area due to demography, environmental exposures, and healthcare practices. Due to variances in case definitions, diagnostic criteria, and research techniques, ILD prevalence estimates are difficult to calculate, but known data show it is a global public health issue. European, North American, and Asian studies have shown ILD prevalence rates of 10 to 40 cases per 100,000 population, with greater rates in older age groups and particular occupational or environmental contexts.

Idiopathic pulmonary fibrosis (IPF), the most prevalent and well-studied form of ILD, with a median survival time of 2 to 5 years following diagnosis and an annual incidence of 2 to 29 cases per 100,000 population. Most

IPF instances occur in persons over 50. IPF's aetiology is unclear, however smoking, environmental exposures, and genetic predispositions are risk factors. Rheumatoid arthritis, systemic sclerosis, and systemic lupus erythematosus may cause connective tissue disease-associated ILD (CTD-ILD), another important subtype of ILD. ILD affects up to 30% of connective tissue disease patients, with prevalence rates varying by autoimmune illness [17]. ILD is seen in 5–10% of rheumatoid arthritis patients and 40–90% of systemic sclerosis patients. Hypersensitivity pneumonitis (HP) is an ILD induced by an overreaction to environmental antigens such mould spores, bird droppings, and occupational dusts. Epidemiological studies have linked HP to occupational exposures in farming, animal husbandry, and carpentry. Familial clustering of HP cases suggests a hereditary propensity. Sarcoidosis is another ILD with epidemiological variations in frequency, age distribution, and race. Sarcoidosis is more common among 20–40-year-olds. Sarcoidosis is also more common among African-Americans than Caucasians.

3. Clinical Assessment in Interstitial Lung Disease (ILD)

Clinical assessment helps evaluate and treat interstitial lung disease (ILD) patients by revealing illness presentation, progression, and prognosis. ILD is a broad collection of parenchymal lung illnesses that cause lung interstitium inflammation and fibrosis. Its symptoms and causes vary. A thorough history-taking, physical examination, and comorbidity assessment identifies ILD subtypes, guides diagnostic tests, and optimises patient therapy. ILD symptoms vary and may include respiratory and extrapulmonary symptoms [18]. ILD is characterised by exertional dyspnea, which commonly precedes non-productive cough, tiredness, and exercise intolerance. Dyspnea severity and progression depend on ILD subtype, illness stage, and comorbidities. Extrapulmonary symptoms as digital clubbing, skin rash, and joint pain may indicate a systemic illness or environmental exposure in ILD patients.

A detailed history is needed to identify ILD risk, trigger, and aggravating variables. ILD evaluation often considers occupational exposures to inhaled dusts, chemicals, or environmental toxins, especially in farming, mining, construction, and manufacturing. A complete medication history is also important for diagnosing drug-induced ILD, since cytotoxic, anti-inflammatory, and biological therapies have been linked to ILD. Disease severity and consequences affect ILD patients' physical examination results. Fine end-inspiratory crackles on auscultation indicate interstitial inflammation and fibrosis. During exacerbations or acute respiratory decompensation, ILD patients may have tachypnea, auxiliary muscle usage, and cyanosis. Some ILD subtypes include extrapulmonary signs such digital clubbing, cutaneous telangiectasia, or joint abnormalities, which might aid diagnosis. The thorough examination of ILD patients must include comorbidities, which may affect disease progression, treatment response, and prognosis. GERD, pulmonary hypertension, coronary artery disease, and obstructive sleep apnea are common ILD comorbidities. ILD may occur with autoimmune illnesses such rheumatoid arthritis, systemic sclerosis, and systemic lupus erythematosus, requiring a multidisciplinary approach to diagnosis and treatment [19]. The figure 2 highlights key characteristics of Interstitial Lung Disease (ILD) through a schematic illustration, offering a visual representation of the complex pulmonary pathology for enhanced understanding and diagnostic clarity.

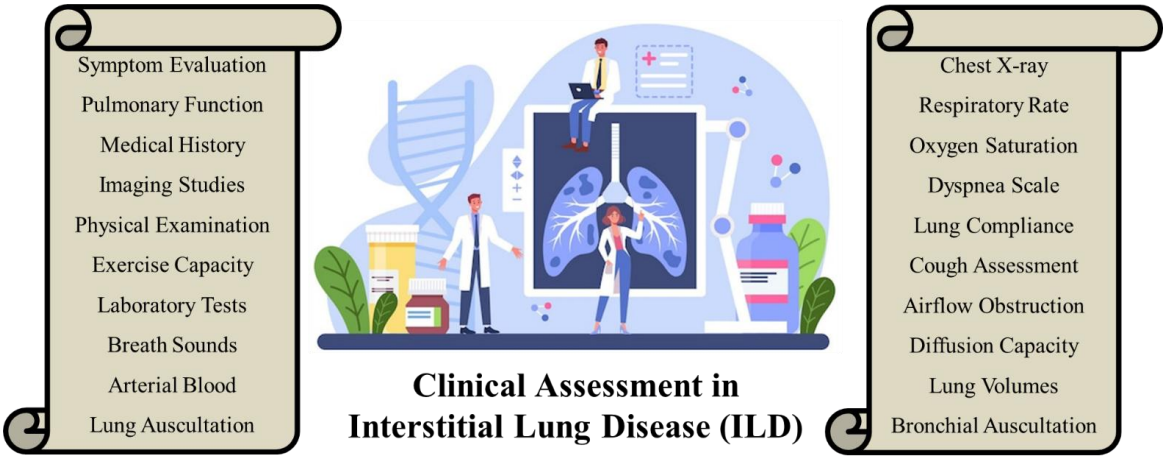

Figure 2. Comprehensive Clinical Assessment in Interstitial Lung Disease

3.1 Presenting Symptoms in Interstitial Lung Disease (ILD)

Interstitial lung disease is a heterogeneous collection of parenchymal lung illnesses that cause lung interstitium inflammation and fibrosis. ILD symptoms are generally vague and vary by aetiology, illness severity, and patient features. These symptoms must be identified quickly for diagnosis and treatment. The most prevalent and severe symptom of ILD is dyspnea. As the condition advances, exertional dyspnea may start mildly but worsen. Climbing stairs, walking, and doing chores may cause respiratory problems for patients [20]. ILD parenchymal fibrosis and inflammation limit gas exchange and lung compliance, causing dyspnea. Another common symptom of ILD is non-productive cough, which may vary in intensity. Dry, persistent coughs without sputum or respiratory illnesses are common. Inflammatory mediators or fibrotic alterations may cause cough in ILD.

ILD patients often experience fatigue and malaise, which may lower quality of life and function. ILD fatigue may be caused by prolonged hypoxemia, sleep problems, deconditioning, or comorbidities. Even after little exercise, patients may feel fatigued or drained. ILD patients with severe fibrosis or acute exacerbations may have chest pain. Pleuritic discomfort, which intensifies with inspiration or movement, may be localised to the chest wall or substernum. ILD may cause chest discomfort due to mechanical pressure on the pleura, lung parenchyma inflammation, or underlying comorbidities such pulmonary hypertension or heart illness. Digital clubbing bulbous fingers and nails is a unique physical feature in certain ILD patients. Chronic hypoxemia and tissue hypoxia cause clubbing by remodelling distal phalangeal vascular tissue and proliferating connective tissue. Clubbing is not unique to ILD and may occur in other pulmonary and cardiac diseases, although it should warrant lung pathology examination [21]. Immune problems or environmental exposures may cause extrapulmonary symptoms such joint pain, skin rash, or systemic symptoms in ILD patients. ILD-CTD overlap syndromes may develop in connective tissue illnesses include rheumatoid arthritis, systemic sclerosis, and dermatomyositis. These people may have arthritis, skin abnormalities, or muscular weakness due to their autoimmune illness.

3.2 Physical Examination Findings in Interstitial Lung Disease (ILD)

Interstitial lung disease (ILD) is a heterogeneous collection of parenchymal lung illnesses that cause lung interstitium inflammation and fibrosis. Clinical history, radiological imaging, and pulmonary function tests are used to diagnose ILD, but physical examination results may reveal disease severity, progression, and systemic symptoms. A thorough physical exam helps doctors detect ILD symptoms and guide treatment [22]. ILD respiratory examination typically shows mild but severe lung disease. Velcro crackles, caused by compressed alveoli and distal airways opening suddenly after inspiration, may be heard on lung auscultation. The strength of these bilateral, basal, diffuse crackles depends on disease severity. Velcro crackles are a characteristic of ILD and indicate interstitial fibrosis or alveolitis.

In certain ILD patients, digital clubbing bulbous fingers and nails is a distinguishing physical symptom. Chronic hypoxemia and tissue hypoxia cause clubbing by remodelling distal phalangeal vascular tissue and proliferating connective tissue. Clubbing is not unique to ILD and may occur in other pulmonary and cardiac illnesses, although it should urge additional investigation for lung pathology and systemic diseases. In ILD patients with severe hypoxemia and poor gas exchange, cyanosis bluish skin and mucous membranes may develop [22]. Cyanosis in the lips, nails, and peripheral extremities implies respiratory insufficiency-induced tissue hypoxia. It indicates severe illness and requires immediate examination and treatment to enhance oxygenation and avoid consequences. ILD patients may experience respiratory distress during acute exacerbations or respiratory compromise, characterised by increased labour of breathing, auxiliary muscle usage, or paradoxical chest wall movement. To improve lung mechanics and reduce dyspnea, patients may lean forward with supported arms in a tripod posture. Respiratory distress indicates respiratory failure and needs rapid medical intervention, including oxygen and ventilatory assistance. ILD patients may have extrapulmonary symptoms on physical examination, especially if they have autoimmune illnesses or environmental exposures. ILD-CTD overlap syndromes may cause musculoskeletal symptoms such joint pain, stiffness, and edoema, suggesting rheumatologic illness [23]. Erythema, sclerosis, and telangiectasia may develop in ILD patients with systemic sclerosis or dermatomyositis. Pulmonary hypertension, a typical consequence of severe ILD, may cause right heart strain on cardiovascular testing. Jugular venous distension, hepatojugular reflux, and lower extremity edoema may suggest right ventricular dysfunction and pulmonary vascular resistance. Right-sided heart failure, tricuspid regurgitation murmur, and pulmonary component of the second heart sound (P2) may also be found on auscultation.

3.3 Comorbidities in Interstitial Lung Disease (ILD)

Many medical disorders may affect interstitial lung disease (ILD) development, treatment response, and patient outcomes. Comprehensive ILD care requires understanding the prevalence, impact, and treatment of various comorbidities, which may impair prognosis and quality of life. Gastrosophageal reflux disease (GERD), which causes stomach contents to flow retrograde into the oesophagus, is a common ILD comorbidity. GERD is frequent

in IPF and other fibrosing ILD patients and may contribute to disease progression and exacerbations. Chronic gastric microaspiration may worsen ILD pathophysiology by causing lung damage, inflammation, and fibrosis [24]. For ILD patients with GERD, lifestyle changes, proton pump inhibitors, and antireflux medications decrease symptoms and avoid consequences. Another major complication of ILD is pulmonary hypertension (PH), especially in late stages. PH causes right ventricular dysfunction and heart failure due to high pulmonary arterial pressure and vascular resistance. ILD patients typically develop PH due to prolonged hypoxemia, vascular remodelling, and lung parenchyma fibrosis. PH increases ILD morbidity and mortality and may need vasodilator therapy and supplementary oxygen to enhance oxygenation and hemodynamics.

ILD patients often have obstructive sleep apnea (OSA), which may worsen respiratory symptoms and sleep difficulties. Recurrent upper airway collapse during sleep causes OSA, hypoxia, hypercapnia, and sleep fragmentation. ILD OSA patients may report worsened dyspnea, tiredness, and daytime drowsiness, reducing quality of life and function. ILD OSA treatment frequently requires CPAP therapy to preserve airway patency and promote nocturnal oxygenation. ILD is often linked to autoimmune diseases including RA, SSc, and DM, which may contribute to disease aetiology and clinical symptoms. ILD-CTD overlap syndromes, which include ILD and autoimmune characteristics, are difficult to diagnose and treat. In addition to ILD therapy, these patients may need immunosuppressive medicines to control systemic inflammation and avoid lung harm. Cardiovascular comorbidities such coronary artery disease, heart failure, and arrhythmias are common in ILD patients and might complicate treatment and prognosis [24]. ILD's chronic hypoxemia, pulmonary hypertension, and systemic inflammation raise cardiovascular risk and unfavourable cardiac events. In ILD patients, complete cardiovascular examination and risk stratification are necessary to optimise cardiovascular health and avoid problems. COPD is another significant comorbidity in ILD patients, especially smokers and environmental exposures. Dyspnea and cough are common respiratory symptoms in COPD and ILD, making diagnosis difficult. ILD patients with COPD may have greater respiratory symptoms and functional impairment, requiring specialised therapy.

4. Radiological Evaluation in Interstitial Lung Disease (ILD)

Radiological assessment is crucial for diagnosing, classifying, and monitoring interstitial lung disease. High-resolution computed tomography (HRCT) is best for assessing ILD because to its high spatial resolution and ability to detect minor parenchymal abnormalities. HRCT data on lung involvement distribution, degree, and pattern help ILD patients differentiate diagnosis and choose therapy. ILD is characterised by linear or curvilinear reticular opacities in the lung parenchyma. Idiopathic pulmonary fibrosis (IPF) and other fibrosing ILDs often have reticular opacities caused by lung interstitium fibrotic alterations and thickening. HRCT reticular opacities frequently indicate chronicity and permanent fibrotic alterations, emphasising the significance of early identification and intervention in ILD therapy [20]. Another typical radiographic finding in ILD is ground-glass opacities (GGOs), which are hazy or transparent patches of increased lung attenuation without obscuring bronchial structures or arteries. In ILD subgroups such nonspecific interstitial pneumonia (NSIP), organising pneumonia (OP), and hypersensitivity pneumonitis, GGOs indicate alveolar inflammation, edoema, or partial airspace filling. HRCT GGO distribution and extent assist diagnose ILD entities. Table 2 delineates key radiological findings in Interstitial Lung Disease (ILD), elucidating characteristic imaging features for enhanced diagnostic interpretation and clinical decision-making.

Table 2. Imaging Results of the Radiological Evaluation of Interstitial Lung Disease

Radiological Finding	Description
Reticular Opacities	Linear or curvilinear opacities extending through lung parenchyma, indicative of interstitial fibrosis.
Ground-Glass Opacities	Hazy areas of increased lung attenuation without obscuration of underlying structures, reflecting alveolar inflammation.
Honeycombing	Cystic airspaces with thick walls and intervening fibrosis, indicative of advanced fibrotic remodeling.
Traction Bronchiectasis	Dilatation and elongation of bronchi due to surrounding fibrotic changes, reflecting lung parenchymal distortion.
Distribution Patterns	Peripheral and subpleural distribution with basal predominance is characteristic of idiopathic pulmonary fibrosis.

Honeycombing, a radiological characteristic of advanced fibrotic ILDs such idiopathic pulmonary fibrosis (IPF), is end-stage lung remodelling with cystic airspaces with thick walls and intervening fibrosis. Honeycombing in the subpleural and basal lungs causes permanent parenchymal damage and poor prognosis in ILD patients. Honeycombing on HRCT is a prognostic indication and may impact treatment options, including lung transplantation in advanced instances. Another common radiographic feature in ILD is traction bronchiectasis, which dilates and elongates bronchi owing to fibrotic alterations. Traction bronchiectasis is caused by fibrotic bands mechanically distorting lung parenchyma and revealing interstitial fibrosis and architectural disturbance. Traction bronchiectasis on HRCT correlates with ILD disease severity, progression, and prognosis, making it a helpful biomarker for disease surveillance and therapy response evaluation. HRCT parenchymal abnormality distribution patterns help diagnose ILD subtypes and aetiologies. Idiopathic pulmonary fibrosis (IPF) has peripheral and subpleural fibrotic alterations with basal predominance, whereas hypersensitivity pneumonitis (HP) has central and peribronchovascular distribution with upper lobe predominance. These distribution patterns help restrict ILD diagnoses and guide examination.

4.1 High-Resolution Computed Tomography (HRCT)

HRCT is essential for diagnosing and treating interstitial lung disease (ILD) because it provides unparalleled insights into lung parenchymal architecture with high spatial resolution. HRCT scans tiny sections (≤1 mm) using high-resolution detectors on specialist CT scanners. This method improves spatial resolution, allowing precise visualisation of ILD-related parenchymal anomalies [25]. HRCT's ability to discern fine anatomical features and pathological alterations in the lung interstitium allows for precise ILD subtyping and treatment recommendations based on radiological results. HRCT helps diagnose ILD in clinical practice by revealing radiological patterns. Reticular opacities, linear or curvilinear opacities in the lung parenchyma, indicate interstitial fibrosis and scarring, hallmarks of fibrosing ILDs like IPF. Ground-glass opacities (GGOs) are common in ILDs like nonspecific interstitial pneumonia (NSIP), organising pneumonia (OP), and hypersensitivity pneumonitis (HP). They reflect alveolar inflammation, edoema, or partial airspace filling.

HRCT is useful for disease monitoring and longitudinal ILD progression and therapy response. Serial HRCT imaging helps doctors follow radiological trends, identify illness exacerbations, and assess therapy effectiveness. HRCT radiological abnormalities such honeycombing or traction bronchiectasis correlate with disease severity, progression, and prognosis, affecting ILD treatment and patient care. HRCT is useful for diagnosis, however it has drawbacks. Radiation exposure, contrast agent delivery, and patient compliance may affect HRCT imaging quality and safety [25]. Interpreting HRCT data needs knowledge of ILD patterns since modest radiological abnormalities might be difficult to distinguish from normal fluctuations or other lung diseases. HRCT is still essential for evaluating and managing ILD, directing treatment, prognosticating disease outcomes, and optimising patient care. Future advances in HRCT technology and interpretation will improve ILD knowledge and management.

4.2 Chest X-ray Findings

In the diagnosis of interstitial lung disease (ILD), chest X-rays reveal parenchymal abnormalities and guide further investigation. Chest X-ray may suggest ILD and require further imaging and assessment, although it is less sensitive than high-resolution computed tomography (HRCT) in detecting small lung abnormalities [26]. Reticulonodular opacities, tiny linear or nodular densities in the lung fields, are a characteristic of ILD on chest X-ray. These lung parenchyma opacities indicate interstitial thickening and fibrosis, suggesting ILD. HRCT provides more precision and detail than chest X-ray reticulonodular opacities, making it difficult to distinguish ILD subtypes. In ILD patients, chest X-rays may show ground-glass opacities (GGOs), which are less prevalent than HRCT. GGOs appear as hazy or weak lung opacities without bronchial structures or arteries. GGOs may indicate alveolar inflammation or interstitial edoema in ILD, while conventional chest X-rays have poorer sensitivity.

In advanced ILD, chest X-rays may show lung parenchyma fibrotic remodelling and architectural deformation. Honeycombing clustered cystic airspaces with thick walls and fibrosis is one of these findings. Advanced fibrotic ILDs such idiopathic pulmonary fibrosis cause honeycombing in the subpleural and basal lungs [26]. Chest X-rays can identify honeycombing, however HRCT can better characterise it. Chest X-rays of ILD patients may show lung volume decrease, pleural thickening, and reticular reticulation. Radiographic abnormalities are vague and may be seen in many lung diseases, thus clinical history, physical examination, and HRCT should be correlated.

4.3 Other Imaging Modalities

In addition to chest X-ray and HRCT, several other imaging modalities help diagnose, monitor, and treat interstitial lung disease (ILD). Magnetic resonance imaging (MRI), positron emission tomography (PET) and lung ultrasound each have their own benefits for ILD evaluation. Magnetic resonance imaging is becoming a useful supplementary imaging technique for ILD assessment, especially where radiation exposure or contrast agent administration must be minimised. Without ionising radiation, MRI can see lung parenchyma and anomalies due to its soft tissue contrast and multiplanar imaging. HRCT is the gold standard for ILD imaging, however MRI may help detect mediastinal or pleural involvement, focal lesions, disease activity, and therapeutic response. PET/CT/MRI is a useful method for monitoring disease activity, inflammation, and metabolic alterations in ILD [27]. PET imaging detects lung parenchyma glucose metabolism and cellular activity using radiolabeled tracers like fluorodeoxyglucose (FDG). In ILD, PET imaging can distinguish active inflammatory processes from fibrotic alterations, guide biopsy site selection, and track therapy response. PET imaging results should be evaluated alongside clinical and radiographic data since infectious or neoplastic diseases may enhance FDG uptake.

The rapidly growing lung ultrasound (LUS) imaging method provides real-time, radiation-free lung parenchyma and pleural space evaluation. A-lines (horizontal reverberation artefacts) and B-lines (vertical comet tail artefacts) represent air-filled or fluid-filled lung spaces, respectively, and are visualised using high-frequency ultrasound waves. ILD patients' pleural effusions, pneumothorax, and consolidations may be assessed immediately at the bedside with LUS for diagnostic and therapeutic advice. LUS may be done serially to track disease development and therapy response, supplementing standard imaging. Each imaging technique has pros and cons, but a multimodal imaging strategy customised to patient requirements and clinical context is frequently best for ILD assessment. Lung parenchyma, pleural space, and anomalies may be assessed by chest X-ray, HRCT, MRI, PET, and LUS, enabling accurate diagnosis, disease staging, and therapy planning in ILD patients. Further advances in imaging technology and methodologies will improve ILD evaluation and management, increasing patient outcomes and quality of life.

5. Pulmonary Function Testing in ILD

Pulmonary function testing (PFT) helps diagnose and treat interstitial lung disease (ILD) by revealing lung mechanics, gas exchange, and respiratory function. PFT enables doctors analyse lung volumes, capacities, and gas diffusion to determine disease severity, monitor progression, and guide treatment in ILD patients. The main goal of PFT in ILD is to measure lung volumes, including total lung capacity (TLC), vital capacity (VC), and residual volume (RV). VC measures the maximum forceful exhalation following a maximal inhalation, whereas TLC measures the highest volume of air in the lungs after a maximal inspiration. Fibrotic alterations and decreased lung parenchyma compliance lower TLC and VC in ILD, causing restrictive ventilatory impairment [20]. PFT usually shows a restrictive ventilatory defect with reduced TLC and VC in idiopathic pulmonary fibrosis (IPF), a progressive ILD. PFT also measures gas exchange and diffusion capacity, such as the lung's carbon monoxide diffusing capacity (DLCO) and alveolar-arterial oxygen gradient (A-a gradient). DLCO indicates gas exchange efficiency in ILD by measuring the lung's capacity to move gas from inspired air to the bloodstream via the alveolar-capillary membrane. ILDs with severe fibrosis and alveolar injury, such as IPF or sarcoidosis, have lower DLCO owing to decreased gas exchange surface area and thickening of the alveolar-capillary barrier. Table 3 offers a comprehensive overview of essential pulmonary function testing parameters commonly evaluated in individuals with Interstitial Lung Disease, aiding in the assessment and management of respiratory function.

Table 3. Overview of Pulmonary Function Testing Parameters in Interstitial Lung Disease

Pulmonary Function Test	Description	Example
Forced Vital Capacity (FVC)	The total volume of air exhaled forcefully and maximally after a maximal inhalation, reflects lung capacity and respiratory effort.	Measurement: 4.5 liters
Forced Expiratory Volume in 1 second (FEV1)	The volume of air forcibly exhaled in the first second of the FVC maneuver, reflects the rate of airflow through the airways.	Measurement: 3.8 liters
FEV1/FVC Ratio	The ratio of FEV1 to FVC, used to assess for airflow obstruction, with reduced values indicative of airway narrowing or obstruction.	Calculation: 3.8 L / 4.5 L = 0.84

Total Lung Capacity (TLC)	The maximum volume of air contained in the lungs at the end of a maximal inspiration, reflects lung volume and respiratory effort.	Measurement: 6.0 liters
Diffusing Capacity of the Lung for Carbon Monoxide (DLCO)	Reflects the ability of the lungs to transfer gas from inspired air to the bloodstream across the alveolar-capillary membrane.	Measurement: 70% of predicted value
Inspiratory Reserve Volume (IRV)	The additional volume of air that can be inhaled beyond a normal tidal breath, reflects lung capacity and respiratory effort.	Measurement: 1.0 liters

PFT evaluates lung volumes, gas exchange, airflow obstruction, and bronchial hyperreactivity in ILD patients to detect airway involvement or comorbidities. FEV1 and FEV1/FVC ratios detect airflow restriction and distinguish restrictive and obstructive ventilatory patterns. While ILD is predominantly a restrictive ventilatory condition, concurrent airway disorders like asthma or COPD may affect airflow dynamics and worsen respiratory symptoms. Serial PFTs can help ILD patients track disease progression and medication response. Clinicians may monitor disease stability, identify exacerbations, and evaluate treatment measures like pharmaceutical drugs or pulmonary rehabilitation by comparing longitudinal lung function metrics. In patients with ILD undergoing antifibrotic medication for IPF, repeated PFTs are used to monitor lung function decrease and evaluate treatment response based on lung volumes, gas exchange, and symptomatology.

5.1 Spirometry

Spirometry is a basic pulmonary function test (PFT) used to diagnose interstitial lung disease (ILD) by measuring airflow dynamics and lung volumes. Spirometry helps diagnose, classify, and monitor ILD by measuring FVC, FEV1, and the FEV1/FVC ratio [28]. Spirometry involves maximum inhaling and quick, forceful expiration into a spirometer. Spirometric parameters are calculated by measuring the volume and flow rate of air evacuated from the lungs during the manoeuvre. FVC measures lung capacity and respiratory effort by measuring the total volume of air forcibly expelled from maximum inhalation to maximum exhalation. FEV1 is the volume of air forced out in the first second of the FVC manoeuvre and indicates airway airflow. The FEV1/FVC ratio is then computed to determine airflow restriction. Low values indicate airway constriction or blockage, as in COPD or asthma.
Spirometry can assess restrictive ventilatory patterns, which refer to lower lung volumes and restricted lung expansion owing to fibrotic lung parenchyma, in ILD. ILD patients had lower FVC and FEV1, indicating poor lung compliance and mechanics. Spirometry frequently shows a restrictive ventilatory defect with decreased FVC and preserved or slightly reduced FEV1/FVC ratio in idiopathic pulmonary fibrosis (IPF), a progressive fibrosing ILD. In ILD patients, a low DLCO and restricted spirometric patterns indicate poor gas exchange efficiency and parenchymal lung disease [28]. In ILD patients, repeated spirometry is essential for disease progression and medication response monitoring. Clinicians may monitor disease stability, identify exacerbations, and evaluate therapeutic treatments like pharmaceutical drugs or pulmonary rehabilitation by comparing longitudinal spirometric characteristics. In patients with ILD undergoing antifibrotic medication for IPF, serial spirometry is used to monitor lung function decrease and evaluate treatment response based on FVC, FEV1, and symptomatology.

5.2 Lung Volumes and Capacity

Pulmonary function testing (PFT) measures lung volumes and capabilities, which are critical to diagnosing and treating interstitial lung disease. These parameters help diagnose, classify, and monitor ILD by revealing respiratory mechanics, lung compliance, and gas exchange efficiency. Total lung capacity (TLC) is the greatest air volume in the lungs after a maximal inhalation. TLC includes inspiratory reserve volume (IRV), tidal volume (TV), expiratory reserve volume (ERV), and residual volume [29]. IRV is the extra air that may be taken beyond a regular tidal breath, whereas ERV is the strong exhalation following one. RV, the volume of air left in the lungs after maximum exhalation, cannot be determined by spirometry. Fibrotic alterations and lower lung compliance may limit TLC in ILD, affecting ventilatory function and lung expansion.
Lung volumes also determine vital capacity (VC), the highest forceful exhalation following a maximal inhale. The total of tidal, inspiratory, and expiratory reserve volumes is VC, which measures lung capacity and respiratory effort. Fibrous alterations and poor lung compliance limit VC in ILD. Idiopathic pulmonary fibrosis (IPF), a progressive ILD, reduces VC due to decreased lung expansion and restricted lung mechanics. The amount of air

left in the lungs after a typical tidal breath is called functional residual capacity (FRC) and is made up of ERV and RV. FRC indicates lung compliance and gas exchange efficiency, and it changes in several respiratory diseases, including ILD [29]. ILD patients may have higher FRC owing to poor lung compliance and gas exchange, causing ventilation-perfusion mismatch and hypoxemia. In ILDs with significant fibrosis and alveolar destruction, such as IPF or sarcoidosis, poor lung compliance and inefficient gas exchange may raise FRC.

5.3 Diffusion Capacity

Lung diffusing capacity for carbon monoxide (DLCO) is a key metric in pulmonary function testing (PFT) for interstitial lung disease (ILD). DLCO measures the lungs' capacity to move gas from inspired air to the bloodstream via the alveolar-capillary membrane, revealing gas exchange efficiency and function [30]. DLCO is measured by inhaling a tiny amount of known carbon monoxide (CO) and holding for a short time. CO diffuses past the alveolar membrane into the circulation and binds to red blood cell haemoglobin. Blood CO uptake is directly related to gas exchange surface area and inversely proportional to alveolar-capillary barrier thickness.
DLCO is computed as a percentage of projected normal values by comparing CO concentrations in exhaled breath and inspired gas. A sensitive measure of parenchymal lung disease severity and gas exchange impairment in ILD is DLCO. Due to fibrotic alterations, alveolar damage, and reduced gas exchange surface area, ILD patients have lower DLCO values. Idiopathic pulmonary fibrosis (IPF), a progressive fibrosing ILD with substantial lung parenchyma remodelling, decreases DLCO owing to alveolar surface area loss and alveolar-capillary membrane thickening [30]. Sarcoidosis, an inflammatory ILD with granulomatous infiltrates, may lower DLCO due to alveolar inflammation and gas diffusion. In ILD patients, serial DLCO monitoring is essential for disease progression and therapy response. Clinicians may monitor disease stability, identify exacerbations, and evaluate therapeutic measures like pharmaceutical drugs or pulmonary rehabilitation by evaluating longitudinal DLCO changes. Patients with ILD taking antifibrotic medication for IPF are regularly measured for serial DLCO to monitor gas exchange efficiency and evaluate treatment response based on DLCO and symptomatology.

2. Conclusion

The multifarious character of interstitial lung disease (ILD) may be better understood by comparing and correlating clinical, radiographic, and pulmonary function testing in tertiary care patients. Clinicians may improve diagnosis accuracy, illness categorization, and patient therapy by integrating clinical, imaging, and functional data. Such studies demonstrate the variability of ILD presentations and the need for comprehensive disease care. If researchers compare clinical features, high-resolution computed tomography (HRCT), and pulmonary function test results in patients with different ILD subtypes, they may find radiological abnormalities that match clinical phenotypes. This association allows for phenotype-driven therapy methods, such as antifibrotic medicines for progressive fibrosis in fibrotic ILDs and immunosuppressive medications for inflammatory ILDs.
Prognostic information from radiological and pulmonary function test correlations aids risk categorization and treatment decisions. In patients with idiopathic pulmonary fibrosis (IPF), a progressive fibrosing ILD, HRCT fibrotic changes and pulmonary function tests like FVC and DLCO are strong predictors of disease progression and mortality. Thus, combining radiological and functional data identifies people at greater risk of illness development who may benefit from early intervention or clinical trials. Comparative research also improves ILD diagnosis criteria and classification systems. By understanding the association between clinical, radiological, and functional characteristics, researchers may develop new diagnostic criteria or improve classification methods to better stratify patients by illness phenotype, prognosis, and treatment response. This optimises therapeutic actions and reduces treatment-related side effects, enabling personalised medicine and improving patient outcomes. Clinical, radiographic, and pulmonary function testing in ILD patients are essential to evidence-based medicine, revealing disease aetiology, prognosis, and therapy response. Clinicians and researchers may improve patient treatment and outcomes by collaborating and conducting comprehensive comparative studies to better understand ILD.

3. References

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