
Comparison of HRCT Findings in Interstitial Lung Disease among Diabetic and Non-Diabetic Patients

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Abstract

Background: Diabetes mellitus (DM) is a chronic metabolic disease and its prevalence has been steadily increasing all over the world. DM and its associated micro and macrovascular complications result in significant morbidity and mortality. The microvascular complications are usually manifested as retinopathy, neuropathy, nephropathy and macrovascular complications generally affect the cardiovascular system. In addition to these complications, DM also affects the lungs because of its rich vascularity and abundance in connective tissue (collagen and elastin). DM has been found to cause microvascular complications and proliferation of extracellular connective tissue in the lungs, leading to decline in lung function in a restrictive pattern of Interstitial lung disease (ILD).

Objective: To compare HRCT patterns and severity of interstitial lung diseases in diabetic and non-diabetic patients.

Methods: A comparative cross-sectional study was conducted on patients diagnosed with interstitial lung disease, dividing them into diabetic and non-diabetic groups. All participants underwent High-Resolution Computed Tomography (HRCT) of the chest, and radiological patterns such as ground-glass opacities, reticulation, and honeycombing were systematically evaluated. The HRCT findings were then compared between both groups to assess the influence of diabetes mellitus on the pattern and severity of interstitial lung disease.

Results: Furthermore, these findings are consistent with earlier research suggesting that radiological patterns in interstitial lung disease (ILD) are more closely related to disease-specific pathophysiological mechanisms rather than systemic metabolic conditions such as diabetes. Although some studies have reported reduced lung volumes and diffusing capacity in diabetic individuals, these functional impairments may not necessarily correspond to detectable structural abnormalities on imaging. In the present study, no statistically significant association was observed between diabetes and HRCT patterns of ILD. This lack of significance may be partly explained by unassessed variables that could influence pulmonary involvement, including the duration of diabetes, level of glycemic control, presence of diabetic complications, and medication use. The

heterogeneity within the diabetic group may therefore have contributed to the absence of a significant correlation.

Conclusion: The study shows that diabetic and non-diabetic patients were demographically comparable, with no significant variation in ground-glass opacities, nodules, consolidation, reticulations, or other radiological features. Fibrotic, cystic, inflammatory, and atelectatic changes, as well as honeycombing and usual interstitial pneumonia patterns, were similarly distributed in both groups. The extent of lung involvement and prevalence of bronchiectasis were also alike.

Keywords: Diabetes mellitus, Interstitial lung disease, Idiopathic pulmonary fibrosis, Metformin, Diabetes complications, Pulmonary function test, Restrictive lung disease, Hyperglycemia

Introduction

The lung interstitium, which comprises the tissue and space surrounding the alveoli, is the primary site of interstitial lung disease (ILD), a diverse set of chronic and progressive pulmonary illnesses [1]. The interstitium is essential for preserving the lungs' structural integrity and facilitating gas exchange. Thickening of the alveolar walls and disturbance of normal lung architecture are the results of inflammatory processes and fibrotic alterations that take place within this sensitive structure in ILD [2]. These pathological changes ultimately result in respiratory impairment by decreasing the effectiveness of oxygen transport from the alveoli into the circulation. A vast range of illnesses with distinct etiologies, clinical manifestations, radiologic findings, and prognoses are included in the disease spectrum. Despite these variations, the majority of ILD types have comparable pathophysiological pathways that include increasing lung parenchymal fibrosis and persistent inflammation [3].

More than 200 different pulmonary conditions are included under the phrase "interstitial lung disease." Idiopathic pulmonary fibrosis (IPF), nonspecific interstitial pneumonia (NSIP), hypersensitivity pneumonitis, ILD linked to connective tissue illness, and occupational lung disorders like silicosis and asbestosis are among the most prevalent categories [4]. Idiopathic pulmonary fibrosis is thought to be the most severe and progressive of them, with a poor prognosis and persistent fibrotic lung remodeling. While occupational lung disorders are caused by long-term exposure to hazardous substances at work, hypersensitivity pneumonitis is caused by repetitive inhalation of organic antigens. Interstitial lung involvement can also result from connective tissue illnesses such rheumatoid arthritis, systemic sclerosis, and systemic lupus erythematosus [5]. Even though the etiology of these illnesses differ, they all eventually result in anatomical alterations in the lung interstitium that lower quality of life and compromise pulmonary function [6]. Because of its high morbidity and mortality, ILD has gained recognition as a public health issue on a global scale. The reported prevalence of ILD has steadily increased during the last few decades. A number of variables, such as better diagnostic methods, more readily available high-resolution imaging modalities, and increasing knowledge of diffuse parenchymal lung disorders among medical professionals, could be responsible for this increase [7]. A prevalence of 20 to 70 cases per 100,000 people has been found by studies carried out in various locales. However, these numbers differ significantly according on environmental factors, occupational exposures, and geographic location. Higher incidence rates are frequently seen in developed nations as a result of extended exposure to industrial chemicals, dust from the workplace, and pollution [8].

There are very few trustworthy epidemiological data about ILD in poor nations like Pakistan. Nonetheless, hospital-based research that is currently available indicates that the prevalence of ILD is between 30 and 40 occurrences per 100,000 people. Idiopathic pulmonary fibrosis seems

to be the most commonly reported kind among the other subtypes [9]. The disease load is thought to be largely influenced by environmental pollution, growing urbanization, and occupational exposure to chemicals and dust. The management of ILD in developing nations may also be made more difficult by delayed diagnosis and restricted access to specialized diagnostic facilities. The overall burden of ILD is anticipated to rise in the upcoming years due to the aging population and the growing prevalence of chronic disorders [10].

Environmental exposures and host-related variables interact intricately in the multifactorial etiology of interstitial lung disease. Exposure to hazardous chemicals, prolonged contact with industrial or agricultural pollutants, and inhalation of organic and inorganic dusts are examples of environmental causes. Antibiotics, antiarrhythmic medicines, and chemotherapeutic treatments have all been linked to the development of ILD [11]. Additionally, immune-mediated inflammation in the lung interstitium can result from autoimmune and connective tissue diseases. In certain instances, interstitial lung injury may also be a result of viral or fungal infections. Certain kinds of ILD are significantly influenced by genetic susceptibility, especially family forms of idiopathic pulmonary fibrosis where mutations in genes linked to telomerase function or surfactant proteins have been found [12].

Since the underlying etiology of ILD is still unclear in a significant number of patients despite major investigation, these cases are classified as idiopathic. The significance of systemic metabolic abnormalities in the onset and progression of ILD has been investigated by researchers in recent years. Among these, diabetes mellitus has garnered special attention since it affects many organ systems and is widely prevalent [13]. Persistent hyperglycemia brought on by deficiencies in insulin secretion, action, or both is a hallmark of diabetes mellitus, a chronic metabolic disease. The kidneys, eyes, nerves, and cardiovascular system are among the organs affected by microvascular and macrovascular problems brought on by long-term diabetes [14].

Diabetes mellitus may potentially affect the structure and function of the lungs, according to new research. Because of their vast capillary and connective tissue network, the lungs are vulnerable to microvascular damage brought on by long-term hyperglycemia [15]. Diabetes can cause microangiopathy, which thickens the capillary basement membrane and lowers pulmonary tissue perfusion. Furthermore, long-term hyperglycemia increases oxidative stress and the production of advanced glycation end products (AGEs), which can harm lung parenchyma structural proteins. Interstitial abnormalities may arise as a result of these processes, which may also change lung elasticity and decrease pulmonary compliance [16].

Another mechanism linking diabetes with interstitial lung disease is chronic low-grade systemic inflammation. Patients with diabetes often exhibit elevated levels of inflammatory cytokines and oxidative stress markers. These inflammatory mediators may stimulate fibroblast activation and excessive deposition of extracellular matrix components within the lung interstitium [17]. Over time, this process leads to progressive fibrosis and irreversible scarring of lung tissue. As a result, diabetic individuals may have an increased susceptibility to developing ILD or experiencing a more rapid progression of existing interstitial abnormalities [18].

Numerous risk factors have been linked to the development of ILD in a number of studies. One of the most important modifiable risk factors is cigarette smoking, which has been closely associated with several types of interstitial lung disease. Lung inflammation and fibrosis can also be caused by prolonged exposure to environmental contaminants, such as industrial chemicals and particulate matter [19]. Familial types of pulmonary fibrosis have been linked to genetic abnormalities that impact telomerase activity, mucin synthesis, or surfactant proteins. Additionally, secondary ILD can result from immune-mediated processes in chronic inflammatory diseases such polymyositis and rheumatoid arthritis.

Male gender and advanced age are known to be significant indicators of ILD [20]. Age-related changes in lung tissue and cumulative environmental exposures are more common in older people, making them more susceptible to fibrosis

Furthermore, it is becoming more well acknowledged that lung interstitial abnormalities may be caused by metabolic comorbidities such as diabetes mellitus, obesity, and metabolic syndrome [21]. Because diabetes and ILD have similar pathophysiological pathways involving inflammation, oxidative stress, and endothelial dysfunction, their interaction is very important.

In order to improve patient outcomes and provide suitable therapy options, early detection and precise assessment of ILD are essential [22]. In the past, patients with suspected lung illness were evaluated with chest radiography as the first imaging modality. However, the sensitivity needed to identify early or subtle interstitial alterations is frequently lacking in conventional radiography. As a result, the gold standard imaging method for assessing interstitial lung disease is High-Resolution Computed Tomography (HRCT) [23]. HRCT provides highly detailed images of lung parenchyma, allowing visualization of fine anatomical structures that cannot be clearly seen on standard chest X-rays.

In order to diagnose, categorize, and treat ILD, HRCT is essential. It enables medical professionals to recognize distinctive radiological patterns including traction bronchiectasis, honeycombing, reticular anomalies, and ground-glass opacities [24]. These imaging results offer important insights into the disease's severity and stage, as well as the most likely underlying subtype of ILD. For instance, ground-glass opacities may indicate inflammatory processes such as nonspecific interstitial pneumonia or hypersensitivity pneumonitis, while honeycombing and subpleural fibrosis are common characteristics of idiopathic pulmonary fibrosis [25].

Determining the degree and distribution of lung disease is another benefit of HRCT. This knowledge is crucial for distinguishing between different forms of ILD and for directing additional diagnostic tests like lung biopsies. In many instances, a clear diagnosis can be made without invasive testing by combining HRCT results with clinical and laboratory data. HRCT is an essential technique in both clinical practice and research settings since it is also frequently used to track the course of disease and assess therapy response [26]. Understanding the possible effects of diabetes mellitus on lung health has become a crucial field of research due to the disease's rising global incidence. Diabetes is known to cause problems with organs like the kidneys, eyes, and cardiovascular system, but little is known about how it affects the anatomy of the lungs [27]. Reduced lung volumes, decreased diffusing capacity, and structural alterations in the lungs of diabetic individuals have been documented in certain investigations. It is still unknown, nevertheless, how much diabetes affects the onset or severity of interstitial lung disease [28].

Because early detection of interstitial alterations may result in better management approaches and better patient outcomes, the potential link between diabetes and ILD is especially clinically significant [29]. Targeted therapies aimed at lowering systemic inflammation and managing hyperglycemia may help decrease the progression of pulmonary fibrosis if diabetes is a contributing factor. Consequently, more investigation is required to elucidate the connection between these two disorders and to pinpoint possible risk factors that can put diabetes individuals at risk for interstitial lung abnormalities [30].

High-Resolution Computed Tomography offers a useful chance to investigate this link more thoroughly. HRCT can be utilized to evaluate lung results between diabetics and non-diabetics with suspected interstitial lung disease since it can identify minor interstitial changes at an early stage [31]. Researchers can determine whether diabetes is linked to more severe or unique radiological signs of ILD by examining HRCT patterns such as ground-glass opacities, reticulation, and honeycombing [32].

In order to ascertain if diabetes mellitus affects the pattern or severity of these radiological abnormalities, the current study evaluates HRCT results in individuals with interstitial lung disease. This study aims to further knowledge of the possible connection between metabolic problems and lung fibrosis by comparing HRCT results between diabetic and non-diabetic patients [33]. The findings of this study have the potential to improve early identification, direct therapeutic management, and ultimately raise the standard of care for individuals with interstitial lung disease [34].

Interstitial lung disease (ILD) causes fibrosis and reduced lung function, while diabetes mellitus (DM) can lead to systemic inflammation and microvascular damage that may also affect the lungs. Although some evidence suggests a link between diabetes and interstitial changes, it remains underexplored. HRCT is the most sensitive tool for detecting ILD, yet few studies have compared HRCT findings between diabetic and non-diabetic patients. This study aims to fill this gap by assessing whether diabetes influences the pattern or severity of ILD on HRCT to support early diagnosis and better management.

Literature Review

Saxena et al. (2025) examined whether exercise ability in individuals with interstitial lung disease (ILD) is independently impacted by high-resolution computed tomography (HRCT) patterns—usual interstitial pneumonia (UIP) versus non-UIP). 139 individuals having complete spirometry, six-minute walk test (6MWT), and HRCT data were included in the study; of them, 56 (40.3%) were classified as UIP and 83 (59.7%) as non-UIP. There were fewer smokers and a greater percentage of females in the non-UIP group. The two groups' mean projected FVC% was comparable ($P = 0.611$), and there were only weak correlations between FVC and 6MWT characteristics ($r = 0.138$ – 0.227). The 6MWT results for UIP and non-UIP patients did not differ statistically significantly after controlling for FVC. Overall, for a similar level of lung function, exercise capacity was comparable between the two groups, indicating that physiological impairment, rather than radiologic pattern, is the primary determinant of functional limitation in ILD [35].

Venkat Rajasurya et al. (2020), diabetes mellitus (DM) is a chronic metabolic disease that is becoming more common worldwide and has a major impact on morbidity and mortality. It is known to result in both macrovascular problems that impact the cardiovascular system and microvascular problems such retinopathy, neuropathy, and nephropathy. In addition to this, DM affects the lungs because of their high connective tissue content and robust vascularity. Restrictive lung dysfunction may result from microvascular damage and excessive lung connective tissue growth. The term "interstitial lung disease" refers to a group of conditions characterized by lung parenchymal fibrosis and inflammation. Idiopathic pulmonary fibrosis is one of the most severe types, with a high death rate and increasing scarring. As IPF worsens, lung function declines and persistent respiratory failure develops. This review examines the relationship between DM, ILD, and especially IPF, with a focus on the lungs as a target organ in DM. A chronic metabolic disease that is becoming more common worldwide, diabetes mellitus (DM) greatly increases morbidity and mortality. It is known to result in both macrovascular problems that impact the cardiovascular system and microvascular problems such retinopathy, neuropathy, and nephropathy. In addition to this, DM affects the lungs because of their high connective tissue content and robust vascularity. Restrictive lung dysfunction may result from microvascular damage and excessive lung connective tissue growth. The term "interstitial lung disease" (ILD) refers to a group of conditions characterized by lung parenchymal fibrosis and inflammation. The most severe of these is idiopathic pulmonary fibrosis (IPF), which has a high death rate and increasing scarring. Declining

lung function and persistent respiratory failure are the outcomes of IPF progression. This review examines the relationship between DM, ILD, and especially IPF and concentrates on the lungs as a target organ in DM [36].

High-resolution computed tomography (HRCT) of the chest is a crucial tool for differentiating idiopathic pulmonary fibrosis (IPF) from other interstitial lung diseases (ILDs), according to Steven D. Nathan et al. (2019). The vocabulary and classification of HRCT interpretations were updated in 2018 with the introduction of new guidelines. This study assessed the differences in physician interpretations between the old and current HRCT guidelines. The kappa statistic, which measures reader concordance, was 0.5 under the previous rules and dropped to 0.38 under the new ones, showing less agreement. Only 15 HRCTs received unanimous consensus under the new classification, compared to 22 under the previous system. Under both approaches, twelve scans were consistently classified as typical interstitial pneumonia (UIP). Under the new paradigm, ten scans that had been classed as possible UIP under the previous guidelines were primarily reclassified as probable UIP [26]. The majority of the 28 UIP cases that were previously inconsistent were now interpreted as alternate diagnoses. Adoption of the revised HRCT recommendations seems to increase interpreter variability overall, and it is unclear how this may affect the diagnosis and treatment of ILD [37].

Hongmei Zhang et al. (2025). Patients with these pulmonary involvements frequently have higher rates of morbidity and mortality. For efficient care, accurate diagnosis and characterisation are consequently crucial. In CTD-related ILD, high-resolution computed tomography (HRCT) is essential for detecting and discriminating lung involvement patterns. The objective of this study was to examine the HRCT signs of different forms of CTD-ILD. The researchers aimed to improve diagnostic accuracy by comparing the imaging properties. Differentiating CTD-ILDs from other interstitial lung illnesses is made easier by an understanding of unique HRCT patterns. The study's overall findings highlight the value of HRCT in enhancing the early detection and treatment of lung illness linked to CTD. [38]

Min Zhu et al. (2025), quantitative methods are being investigated for more accurate evaluation, and high-resolution computed tomography (HRCT) is crucial for diagnosing and tracking connective tissue disease-associated interstitial lung disease (CTD-ILD). In order to assess radiological severity, this study examined dual-energy CT (DECT) scans of 147 patients. Compared to patients with minimal disease, those with severe CTD-ILD had lower lung volumes, higher Zeff values, and higher mean CT numbers (MCTN), all of which were statistically significant ($p < 0.001$). DECT indicators, such as predicted FVC%, predicted DLCO%, and total extent of involvement (TEI), exhibited moderate relationships with lung function indices. MCTN was found to be the greatest discriminator for widespread CTD-ILD by ROC analysis (AUC = 0.901). The Zeff value was found to be an independent risk factor for dyspnea and cough. Additionally, lung volume was significantly associated with the mental health component of the SF-36 quality-of-life score. Overall, DECT proved useful for quantitatively evaluating the severity and clinical impact of CTD-ILD [39].

Venkat Rajasurya et al. (2020), diabetes mellitus (DM) is a chronic metabolic disease that is becoming more common worldwide and is a major cause of morbidity and mortality. It is linked to both macrovascular problems that impact the cardiovascular system and microvascular problems such as retinopathy, neuropathy, and nephropathy. Additionally, because of their extensive arterial network and connective tissue richness, the lungs are also impacted by diabetes. Microvascular injury and increased extracellular matrix deposition are two aspects of pulmonary involvement that result in restrictive lung dysfunction. The term "interstitial lung disease" (ILD) refers to a broad category of conditions marked by lung parenchymal fibrosis and inflammation.

Among these, idiopathic pulmonary fibrosis (IPF) is a severe type that eventually results in respiratory failure, progressive fibrosis, and a high death rate. Because of the lung's high functional reserve, diabetic microangiopathy may not manifest clinically until much later. More study is required to elucidate underlying mechanisms and potential treatments, including the function of metformin, and early screening and enhanced awareness of diabetes-related lung involvement are crucial [40].

Marlies Wijnenbeek, MD et al. (2022), there are about 200 recognized interstitial lung disorders, ranging from extremely rare to quite common. The majority of interstitial lung illnesses are characterized by fibrosis or inflammation in the interstitial space, which mostly results in poor gas exchange, which lowers exercise tolerance, causes dyspnea, and lowers quality of life. Each of the several interstitial lung diseases has a very distinct outcome. While spontaneous reversibility or stabilization can happen in certain situations, respiratory failure and mortality are a sad reality for many individuals with interstitial lung disease, particularly those who have progressive pulmonary fibrosis. The science of interstitial lung disease has made significant strides in the last three years, including the approval of medications to treat interstitial lung disease linked to systemic sclerosis pulmonary hypertension linked to interstitial lung disease, and various types of progressive pulmonary fibrosis. The most common interstitial lung diseases seen in clinical practice are covered in this seminar, along with an update on their epidemiology, pathophysiology, presentation, diagnosis, course, and treatment. Additionally, we summarize the most recent practice-changing guidelines and discuss how advancements have changed the way interstitial lung illnesses with progressive pulmonary fibrosis are classified and treated. An overview of the debates, uncertainties, and potential future paths is provided at the end [41].

Toby M. Maher et al. (2024), interstitial lung disease is a collection of lung conditions characterized by fibrosis and inflammation, which can cause respiratory failure and progressive dyspnea. It affects roughly 650,000 Americans and results in 25,000–30,000 fatalities a year. Idiopathic pulmonary fibrosis (about one-third of cases), hypersensitivity pneumonitis (15%), and ILD associated with connective tissue disease (25%) are common forms. Exertional dyspnea is the primary symptom, and coughing occurs in roughly 30% of cases. ILD subtypes can be identified with a high degree of sensitivity (91%) using CT scans. Lung function decline, particularly a 5% decrease in FVC during a one-year period, doubles the risk of death. Antifibrotic medications such as pirfenidone and nintedanib slow the course of the illness. Immunomodulators like mycophenolate mofetil may help with connective tissue-related ILD. Oxygen treatment and exercise both help reduce symptoms and enhance quality of life. In advanced situations, lung transplantation can greatly increase survival. Late-stage illness frequently results in pulmonary hypertension, which can be improved by therapies like inhaled treprostinil [42].

Li Zhang et al. (2020), type 2 diabetes mellitus exacerbated by interstitial lung abnormalities is frequently underdiagnosed but, if left untreated, can develop into severe pulmonary fibrosis. Compared to ocular examination, chest CT, especially quantitative CT (QCT), offers a more objective and accurate way to identify lung abnormalities. Significantly higher LAA-700 to -200% in patients with T2DM-ILAs indicates more fibrotic changes in lung tissue. On the other hand, decreased total lung volume (TLV), total airway volume (TAV), TAV/TNV ratio, and LAA-950% indicate structural and vascular alterations. These results show a clear pattern of lung damage in those with diabetes. The generally poor or negligible correlation between HbA1c and CT measures suggests that lung damage may not be predicted by glycemic management one. Peripheral vascular index (TAV/TNV) is the best imaging measure for identifying lung alterations associated with type 2 diabetes. Compared to individual density-based measures, it exhibits greater diagnostic accuracy. The best diagnostic performance is obtained by combining an index of several QCT

parameters. Overall findings indicate that T2DM patients are especially susceptible to injury to tiny pulmonary blood arteries [43].

Objectives

To compare HRCT patterns and severity of interstitial lung diseases in diabetic and non-diabetic patients.

Operational Definition

Diabetes Mellitus (DM):

A chronic metabolic disorder diagnosed in patients with fasting blood glucose ≥ 126 mg/dL or on antidiabetic medication [4].

Non-Diabetic Patient:

A patient with normal blood glucose levels and not on any antidiabetic treatment [8].

Interstitial Lung Disease (ILD):

A group of lung disorders characterized by inflammation and/or fibrosis of the lung interstitium, diagnosed based on clinical features, and HRCT findings [12].

High-Resolution Computed Tomography (HRCT):

Imaging modality used to detect and evaluate ILD, with findings such as ground-glass opacities, reticulations, honeycombing, and traction bronchiectasis [14].

HRCT Severity Score:

A semi-quantitative measure of the extent of lung involvement on HRCT, based on the percentage of each lung lobe affected by ILD changes [16].

Comparison Parameter:

The study will assess differences in HRCT patterns, extent, and severity of ILD between diabetic and non-diabetic patient [18].

Methodology

3.1. Research Design: Cross sectional Analytical study

3.2. Clinical Settings: Basheer Hospital, Sialkot

3.3. Sample Size: A total of 60 patients will be included in the study, divided into two groups:

Group A (Diabetic group): 30 patients diagnosed with diabetes mellitus.

Group B (Non-diabetic group): 30 patients without diabetes, matched for age and sex.

Sample size: **60**

This means 60 or more measurements/surveys are needed to have a confidence level of 95% that the real value is within $\pm 5\%$ of the measured/surveyed value.

Confidence Level: ?	95%	▼	
Margin of Error: ?	5	%	
Population Proportion: ?	4	%	Use 50% if not sure
Population Size: ?			Leave blank if unlimited population size.
Calculate  Clear			

Total Sample Size: 60

3.4. Sampling Technique: Convenient sampling technique.

3.5. Duration of Study: 4 months after the approval of synopsis.

3.6. Selection Criteria

3.6.1. Inclusion Criteria

- Patients aged 30–70 years.
- Diagnosed cases of interstitial lung disease confirmed by clinical assessment and HRCT.
- For diabetic group: diagnosed cases of diabetes.
- And complete medical imaging data available

3.6.2. Exclusion Criteria

- Patients with other systemic illnesses (renal, cardiac, thyroid disorders).
- History of smoking-related chronic lung disease.
- Previous lung surgery or malignancy.
- Acute respiratory infections during HRCT assessment.

Equipment:

High-resolution computed tomography (HRCT) scanner with dual-energy capability for quantitative analysis.

Scanning Technique:

A CT scan will be performed using a 128-slice multidetector CT scanner to obtain detailed images of the abdomen. During the procedure, a dual-head power injector will administer a non-ionic iodinated contrast agent through an 18–20 gauge intravenous (IV) cannula. The contrast dose will be approximately 1.5–2.0 mL per kilogram of body weight to enhance visualization of blood vessels and abdominal organs. The scan will include multiple imaging phases to capture different stages of contrast flow. These phases include the arterial phase (about 25–30 seconds) and the portal venous phase (about 60–70 seconds), which is essential for evaluating liver and portal

circulation. A delayed phase (3–5 minutes) may also be performed if additional information is required. The images will be acquired in thin slices of 1 mm or less and later reconstructed in axial, coronal, and sagittal planes using a workstation or PACS system. Radiologists will evaluate the scans for complications such as portal vein thrombosis, splenomegaly (spleen length greater than 13 cm), and ascites, which will be graded as mild, moderate, or severe. Other cirrhosis-related findings, such as varices, will also be assessed. Finally, the collected data will be analyzed statistically using chi-square and correlation tests, and a p-value of less than 0.05 will be considered statistically significant, while a high-resolution CT scanner with dual-energy capability will support more precise quantitative analysis.

3.7.3 Ethical Consideration

The rules and regulations set by the ethical committee of Superior University Lahore will be followed while conducting the research and the rights of the research participants will be respected.

- Written informed consent (attached) will be taken from all the participants.
- All information and data collection will be kept confidential.
- Participants will remain anonymous throughout the study.
- The subjects will be informed that there are no disadvantages or risks on the procedure of the study.
- They will also be informed that they will be free to withdraw at any time during the process of the study.
- There will no risk associated with this research.
- We will do everything we can to protect your privacy. Your identity will not be revealed in any publication resulting from this study.
- Your participation in this research study is voluntary. You may choose not to participate and you may withdraw your consent to participate any time. You will not be penalized in any way should you decide not you participate or to withdraw from this study.

3.8. Data Collection Procedure:

Data will be gathered from patients at Basheer Hospital Sialkot who have been diagnosed with interstitial lung disease (ILD), divided into groups with and without diabetes. Before being included in the study, each subject will be asked for their informed consent. Age, gender, and medical history including whether or not diabetes is present will all be documented. A uniform high-resolution procedure will be used to conduct HRCT scans of the chest. The goal of imaging will be to detect ILD patterns such as honeycombing, reticulations, and ground-glass opacities. The results will be methodically recorded in an organized data sheet. The groups with and without diabetes will be compared. Every piece of information gathered will be safely kept and preserved for future statistical analysis.

3.9. Data Analysis Procedure:

Data analysis for comparing HRCT findings in ILD among diabetic and non-diabetic patients will use SPSS software for data entry and cleaning. Descriptive statistics will summarize demographic details such as age, gender, and disease duration. HRCT patterns, including ground-glass opacities, reticulations, and honeycombing, will be categorized and their frequencies calculated. The chi-square test will compare categorical variables, while t-test or Mann–Whitney U test will analyze continuous kappa statistic. A p-value <0.05 will indicate statistical significance. Results will be displayed in tables for clarity. Conclusions will determine differences in HRCT patterns between diabetic and non-diabetic ILD patients.

Results

Furthermore, these findings are consistent with earlier research suggesting that radiological patterns in interstitial lung disease (ILD) are more closely related to disease-specific pathophysiological mechanisms rather than systemic metabolic conditions such as diabetes. Although some studies have reported reduced lung volumes and diffusing capacity in diabetic individuals, these functional impairments may not necessarily correspond to detectable structural abnormalities on imaging. In the present study, no statistically significant association was observed between diabetes and HRCT patterns of ILD. This lack of significance may be partly explained by unassessed variables that could influence pulmonary involvement, including the duration of diabetes, level of glycemic control, presence of diabetic complications, and medication use. The heterogeneity within the diabetic group may therefore have contributed to the absence of a significant correlation.

Table 4.1 Table of Demographic Characteristics

Variable	Diabetic (n=23)	Non-Diabetic (n=37)	p-value
Age (mean $\pm$ SD)	53.2 $\pm$ 11.5	56.8 $\pm$ 14.2	0.27
Gender (M/F)	12 / 11	22 / 15	0.81

The mean age of diabetic patients (53.2 $\pm$ 11.5 years) was slightly lower than that of non-diabetic patients (56.8 $\pm$ 14.2 years), but this difference was not statistically significant ($p = 0.27$). Gender distribution was also comparable between the groups, with 12 males and 11 females in the diabetic group and 22 males and 15 females in the non-diabetic group, showing no significant difference ($p = 0.81$). Overall, the two groups were demographically similar.

Table 4.2 Table of HRCT Findings in Diabetic vs Non-Diabetic ILD

HRCT Feature	Diabetic (n=23)	Non-Diabetic (n=37)	p-value
Ground-glass opacities	4 (17.4%)	9 (24.3%)	0.52
Nodules	6 (26.1%)	12 (32.4%)	0.61
Consolidation	3 (13.0%)	8 (21.6%)	0.41
Honeycombing	2 (8.7%)	6 (16.2%)	0.38

HRCT Feature	Diabetic (n=23)	Non-Diabetic (n=37)	p-value
Reticulations	5 (21.7%)	10 (27.0%)	0.64
Fibrotic changes	3 (13.0%)	7 (18.9%)	0.58
Cystic changes	2 (8.7%)	5 (13.5% nbbbbbb)	0.56
Inflammatory changes	1 (4.3%)	4 (10.8%)	0.39
Atelactic changes	4 (17.4%)	9 (24.3%)	0.52
Usual interstitial pneumonia	2 (8.7%)	3 (8.1%)	0.94
Frequency			
10			
23			
20			
7			

According to Table 4.2,. The distribution of HRCT features was broadly similar between diabetic and non-diabetic groups, with no statistically significant differences observed for any parameter. Ground-glass opacities, nodules, consolidation, and reticulations were slightly more frequent in non-diabetic patients; however, these differences were not significant ($p > 0.05$). Similarly, fibrotic, cystic, inflammatory, and atelectatic changes showed comparable frequencies between the two groups. Honeycombing and usual interstitial pneumonia patterns were also evenly distributed, with nearly identical proportions. Overall, HRCT findings did not differ significantly between diabetic and non-diabetic patients, suggesting that diabetes status did not influence the radiological pattern of lung involvement in this cohort.

Table 4.3 Table of Lung Involvement

Involvement	Diabetic (n=23)	Non-Diabetic (n=37)	p-value
Unilateral	9 (39.1%)	15 (40.5%)	0.91
Bilateral	14 (60.9%)	22 (59.5%)	—

Regarding table 4.3, The pattern of lung involvement was similar between diabetic and non-

diabetic groups, with unilateral involvement seen in 39.1% of diabetics and 40.5% of non-diabetics ($p = 0.91$), indicating no statistically significant difference. Bilateral involvement was more common in both groups, observed in 60.9% of diabetic patients and 59.5% of non-diabetic patients. Overall, the distribution of unilateral and bilateral disease was comparable between the two groups.

Table 4.4 Bronchiectasis

Bronchiectasis	Diabetic (n=23)	Non-Diabetic (n=37)	p-value
Present	7 (30.4%)	12 (32.4%)	0.87
Absent	16 (69.6%)	25 (67.6%)	—

Table 4.4 The prevalence of bronchiectasis was similar in both groups, being present in 30.4% of diabetic patients and 32.4% of non-diabetic patients, with no statistically significant difference ($p = 0.87$). The majority of patients in both groups did not have bronchiectasis, accounting for 69.6% of diabetics and 67.6% of non-diabetics. Overall, bronchiectasis showed a comparable distribution between diabetic and non-diabetic groups.

Discussion

In order to ascertain whether diabetes mellitus affects the radiological pattern or severity of interstitial lung disease (ILD), the current study assessed and compared HRCT findings of ILD in patients with and without diabetes. Overall, the findings showed no statistically significant variations in HRCT features or demographic variables between the two groups, indicating that diabetes may not independently change the imaging characteristics of ILD in the population under study [44].

The age and gender distribution of the two groups in this study were comparable, which lessens the possibility of confounding and improves the comparison's internal validity. The distribution of HRCT results, such as ground-glass opacities, reticulation, consolidation, honeycombing, and bronchiectasis, was almost identical in individuals with and without diabetes. These results imply that disease-specific fibrotic and inflammatory mechanisms, rather than systemic metabolic state alone, are the main causes of the structural lung alterations observed in ILD [45].

These results demonstrate both agreement and disagreement with earlier research when compared to the body of current literature. As an illustration of a possible biological connection between diabetes and interstitial lung involvement, Venkat Rajasurya et al. (2020) stated that diabetes mellitus can lead to pulmonary microangiopathy, increased connective tissue deposition, and restrictive lung functioning. These theoretical correlations, however, did not appear in the current study's HRCT imaging differences, suggesting that these alterations may continue to be subclinical or undetectable during standard HRCT evaluation [46].

In a similar vein, Ehrlich et al. (2010) proposed that individuals with diabetes have a higher chance

of acquiring idiopathic pulmonary fibrosis, suggesting a potential epidemiological link between diabetes and the development of ILD. However, the prevalence of fibrotic patterns such as honeycombing or UIP did not differ between the diabetic and non-diabetic groups in the current investigation. Variations in study design, sample size, disease severity, or population factors could be the cause of this. However, our results are in line with the study by Saxena et al. (2023), which showed that underlying ILD subtype has a greater impact on disease behavior than comorbidities and that radiological patterns in ILD do not always correlate with functional or systemic conditions [47].

Additionally, Li Zhang et al. (2025) noted that even in cases when conventional imaging appears normal, quantitative CT parameters may identify modest pulmonary vascular and structural alterations in diabetic individuals. This implies that more sophisticated quantitative imaging methods may identify microstructural anomalies that were missed in our analysis, even if routine HRCT in our study did not disclose significant differences [48].

Additionally, there was no discernible difference between the groups in terms of unilateral or bilateral lung involvement. This is consistent with research by Johkoh et al. (2015), who found that patient comorbidities have less of an impact on distribution patterns in ILD than illness type. The hypothesis that chronic airway remodeling in ILD is independent of diabetes state is further supported by the similar prevalence of bronchiectasis in both groups [49].

It's interesting to note that, despite the fact that diabetes is known to induce oxidative stress, systemic inflammation, and the buildup of advanced glycation end products, these processes did not result in discernible radiological variations in this investigation. This may be because pulmonary involvement in diabetes may not be architecturally visible on HRCT, but rather be modest, diffuse, or functionally relevant. It might also mean that any minor alterations in the lungs caused by diabetes are obscured or overshadowed by fibrosis associated with ILD [50].

The fact that this study did not include variables including the length of diabetes, glycemic control (HbA1c levels), the existence of diabetic complications, and medication history is another crucial point. These factors may be crucial in determining the degree of pulmonary involvement, as noted in earlier research, especially by Rajasurya et al. (2020). Their absence may therefore partly explain the lack of significant association observed [51].

Overall, the results of this investigation indicate that the pattern, distribution, and severity of HRCT symptoms of ILD are not substantially affected by diabetes mellitus. This does not, however, totally rule out a biological link between diabetes and lung tissue because traditional imaging methods might not be able to identify minute microvascular or inflammatory alterations [52].

In summary, although earlier research has shown a possible connection between diabetes and interstitial lung abnormalities, our study shows that the HRCT imaging patterns of established ILD do not clearly reflect such a correlation. To better understand this link, more extensive, multi-center research including quantitative imaging, lung function testing, and metabolic control measures is advised [53].

Conclusion

Diabetic and non-diabetic patients were demographically comparable, with no significant differences in age or gender. HRCT findings, including ground-glass opacities, nodules, consolidation, and reticulations, were similar between groups. Fibrotic, cystic, inflammatory, and atelectatic changes, along with honeycombing and usual interstitial pneumonia patterns, showed comparable frequencies. The extent of lung involvement and prevalence of bronchiectasis did not differ significantly. Overall, diabetes did not significantly influence HRCT patterns or disease

extent in interstitial lung disease.

Limitation

The study was limited by a relatively small sample size, which may have reduced the statistical power to detect subtle differences between groups. Being a single-center, cross-sectional study, it also limits generalizability and does not establish causal relationships. Additionally, lack of detailed data on duration and control of diabetes, as well as absence of longitudinal follow-up, may have affected the assessment of its true impact on ILD progression.

Recommendations

Future studies with larger, multi-center populations and longitudinal designs are recommended to better evaluate the relationship between diabetes and ILD progression. Inclusion of parameters such as glycemic control (e.g., HbA1c levels), duration of diabetes, and treatment status could provide deeper insights. Advanced quantitative HRCT analysis and correlation with pulmonary function tests are also suggested to more precisely assess subtle interstitial changes and disease severity

References

- .Hu WS, Lin CL. Effect of anti-diabetic agent on interstitial lung disease in patients with diabetes mellitus. *Naunyn-Schmiedeberg's Archives of Pharmacology*. 2025 Jan;398(1):581-9
- .Kolahian S, Leiss V, Nürnberg B. Diabetic lung disease: fact or fiction?. *Reviews in Endocrine and Metabolic Disorders*. 2019 Sep;20(3):303-19.
- .Zhang L, Fan QJ, Dang S, Han D, Zhang M, Yan SG, Xin XK, Yu N. CT Quantitative Analysis in Evaluating Type 2 Diabetes Mellitus Complicated with Interstitial Lung Abnormalities. *Current Medical Imaging*. 2025 Jan;21(1):E15734056343395.
- Brown KK, Martinez FJ. Clinical course of idiopathic pulmonary fibrosis. *Thorac Soc*. 2010;7(2):178–85.
- Chen L, Zhu M, Lu H, Yang T, Li W, Zhang Y, Xie Q, Li Z, Wan H, Luo F. Quantitative evaluation of disease severity in connective tissue disease-associated interstitial lung disease by dual-energy computed tomography. *Respiratory Research*. 2022 Mar 5;23(1):47. Zhang L, Fan QJ, Dang S, Han D, Zhang M, Yan SG, Xin XK, Yu N. CT Quantitative Analysis in Evaluating Type 2 Diabetes Mellitus Complicated with Interstitial Lung Abnormalities. *Current Medical Imaging*. 2025 Jan;21(1):E15734056343395.
- Classification and diagnosis of diabetes: Standards of medical care in diabetes 2022. *Diabetes Care* 2022;45(1):S1-S17. doi:10.2337/dc22-S00234964875
- Drimus JC, Duma RC, Trăilă D, Mogoșan CD, Manolescu DL, Fira-Mladinescu O. High-resolution CT findings in interstitial lung disease associated with connective tissue diseases: differentiating patterns for clinical practice—A systematic review with meta-analysis. *Journal of Clinical Medicine*. 2025 Aug 31;14(17):6164.
- Ehrlich SF, Quesenberry CP Jr, Van Den Eeden SK, Shan J, Ferrara A. Patients diagnosed with diabetes are at increased risk for idiopathic pulmonary fibrosis. *Diabetes Care*. 2010;33(7):1430–5.
- ELTANTAWY AM, MOHAMMED GE, SAADAWY M. Chest CT Severity Score in COVID-19 Patients: Comparison between Diabetic and Non-Diabetic Patients. *The Medical Journal of Cairo University*. 2022 Dec 1;90(12):2151-60.

- Forgiarini LA, Jr, Kretzmann NA, Porawski M, Dias AS, Marroni NA. Experimental diabetes mellitus: oxidative stress and changes in lung structure. *J Bras Pneumol*. 2009;35:788–791. doi: 10.1590/s1806-37132009000800011
- Hu WS, Lin CL. Effect of anti-diabetic agent on interstitial lung disease in patients with diabetes mellitus. *Naunyn-Schmiedeberg's Archives of Pharmacology*. 2025 Jan;398(1):581–9.
- Johkoh T, Lee KS, Nishino M, Travis WD, Ryu JH, Rosado-de-Christenson ML, et al. Chest CT diagnosis and clinical management of interstitial lung diseases: Part 1. *AJR Am J Roentgenol*. 2015;204(5):941–56.
- Khan NA, Irfan M, Nawaz A, Rizvi N, Haque AS. Spectrum of interstitial lung diseases in Pakistan: A single center study. *Pak J Med Sci*. 2019;35(6):1605–10.
- Kida K, Wakabayashi R, Nishimura M. Diabetes and interstitial lung disease: Mechanistic insights and clinical implications. *Respir Investig*. 2020;58(2):94–101.
- Klein OL, Krishnan JA, Glick S, Smith LJ. Systematic review of the association between lung function and Type 2 diabetes mellitus. *Diabet Med*. 2010;27:977–987. doi: 10.1111/j.1464-5491.2010.03073.x.
- Klein OL, Krishnan JA, Glick S, Smith LJ. Systematic review of the association between lung function and Type 2 diabetes mellitus. *Diabet Med*. 2010;27:977–987. doi: 10.1111/j.1464-5491.2010.03073.x.
- Klein OL, Krishnan JA, Glick S, Smith LJ. Systematic review of the association between lung function and Type 2 diabetes mellitus. *Diabet Med*. 2010;27:977–987. doi: 10.1111/j.1464-5491.2010.03073.x.
- Lederer DJ, Martinez FJ. Idiopathic pulmonary fibrosis. *N Engl J Med*. 2018;378(19):1811–23.
- Li X, Zhang H, Zhang X, Wang G, Zhao X, Zhang J. A comparative study of different types of connective tissue-associated interstitial lung disease. *BMC Medical Imaging*. 2025 Apr 7;25(1):109.
- Lynch DA. HRCT findings of diffuse lung disease: A pattern-based approach. *Radiol Clin North Am*. 2016;54(6):1079–92.
- Marvisi M, Balzarini L, Mancini C, Mouzakiti P, Civardi G, Cuccia MC. Pulmonary involvement in diabetes mellitus: The role of high-resolution computed tomography. *Diabet Med*. 2018;35(12):1706–12.
- Meo SA, Al-Drees AM, Arif M, Al-Rubean K. Lung function in type 2 Saudi diabetic patients. *Saudi Med J*. 2006;27(3):338–43.
- Nalysnyk L, Cid-Ruzafa J, Rotella P, Esser D. Incidence and prevalence of idiopathic pulmonary fibrosis: Review of the literature. *Eur Respir Rev*. 2012;21(126):355–61.
- Nathan SD, Pastre J, Ksovreli I, Barnett S, King C, Aryal S, Ahmad K, Fukuda C, Ramalingam V, Chung JH. HRCT evaluation of patients with interstitial lung disease: comparison of the 2018 and 2011 diagnostic guidelines. *Therapeutic advances in respiratory disease*. 2020 Oct;14:1753466620968496.
- Popov TA, Penev DP. The lung as a target organ in diabetes mellitus. *Diabetes Care*. 2013;36(10):3030–8.
- Prasad N, Veeranki V, Bhadauria D, Kushwaha R, Meyyappan J, Kaul A, Patel M, Behera M, Yachha M, Agrawal V, Jain M. Non-diabetic kidney disease in type 2 diabetes mellitus: A changing spectrum with therapeutic ascendancy. *Journal of clinical medicine*. 2023 Feb 20;12(4):1705.
- Presentation, diagnosis and clinical course of the spectrum of progressive-fibrosing interstitial lung diseases. *Eur Respir Rev*. 2018;27(150):180076.

- Raghu G, Chen SY, Yeh WS, Maroni B, Li Q, Lee YC, et al. Idiopathic pulmonary fibrosis in US Medicare beneficiaries aged 65 years and older: Incidence, prevalence, and survival, 2001–11. *Lancet Respir Med*. 2014;2(7):566–72.
- Rajasurya V, Gunasekaran K, Surani S. Interstitial lung disease and diabetes. *World journal of diabetes*. 2020 Aug 15;11(8):351.
- Rajasurya V, Gunasekaran K, Surani S. Interstitial lung disease and diabetes. *World J Diabetes*. 2020 Aug 15;11(8):351-357. doi: 10.4239/wjd.v11.i8.351. PMID: 32864047; PMCID: PMC7438183.
- Russo A, Patanè V, Oliva A, Viglione V, Franzese L, Forte G, Liakouli V, Perrotta F, Reginelli A. AI-based HRCT quantification in connective tissue disease-associated interstitial lung disease. *Diagnostics*. 2025 Aug 28;15(17):2179.
- Saliba F, Khattar G, Mourad O, Aoun L, Bou Sanayeh E, Arafa F, Al Saidi I, Abidor E, Al Achkar M, Rizvi T, Sangaraju K. Evaluating the impact of type 2 diabetes mellitus on interstitial lung disease prevalence in patients with systemic lupus erythematosus: A national inpatient sample analysis. *Lupus*. 2024 Dec;33(14):1547-55.
- Saliba F, Khattar G, Mourad O, Aoun L, Bou Sanayeh E, Arafa F, Al Saidi I, Abidor E, Al Achkar M, Rizvi T, Sangaraju K. Evaluating the impact of type 2 diabetes mellitus on interstitial lung disease prevalence in patients with systemic lupus erythematosus: A national inpatient sample analysis. *Lupus*. 2024 Dec;33(14):1547-55.
- Saxena P, Singh I, Kumar A, Kartik S, Malik V, Tiwari S, Akhil KR, Pattanayak S, Velangi VG, Jain H. Impact of HRCT pattern on six-minute walk test in patients with interstitial lung disease—An observational study. *Lung India*. 2023 Sep 1;40(5):429-33
- Sgalla G, Iovene B, Calvello M, Ori M, Varone F, Richeldi L. Idiopathic pulmonary fibrosis: Pathogenesis and management. *Respir Res*. 2018;19(1):32.
- Shao C, Xia N, Zhen Y, Zhang X, Yan N, Guo Q. Prognostic significance of natural killer cell depletion in predicting progressive fibrosing interstitial lung disease in idiopathic inflammatory myopathies. *Frontiers in Immunology*. 2024 Apr 30;15:1404828.
- Souza CA, Müller NL, Lee KS, Johkoh T, Mitsuhiro H, Chong S, et al. Idiopathic interstitial pneumonias: Prevalence of high-resolution CT patterns and correlation with clinical findings. *Radiology*. 2003;227(3):695–702.
- Spagnolo P, Rossi G, Cavazza A, Bonifazi M, Paladini I, Bonella F, et al. Hypersensitivity pneumonitis: A comprehensive review. *J Investig Allergol Clin Immunol*. 2015;25(4):237–50.
- Suarez TCG, Contreras E. Increase prevalence of diabetes mellitus in patients with interstitial lung disease. *Am J Respir Crit Care Med*. 2000;161:A829.
- Sverzellati N. Highlights of HRCT imaging in IPF. *Respir Res*. 2013;14(Suppl 1):S3.
- Syed UA, Shravan D, Vishwanath B, Rao VV. Connective Tissue Disorders Associated Interstitial Lung Disease–Evaluation by High Resolution Computed Tomography and Fibrosis Scoring System. *European Journal of Cardiovascular Medicine*. 2025 Jun 9;15:70-5.
- Walsh SLF, Calandriello L, Sverzellati N, Wells AU, Hansell DM. Interobserver agreement for the ATS/ERS/JRS/ALAT criteria for a UIP pattern on CT. *Thorax*. 2016;71(1):45–51.
- Wijsenbeek M, Suzuki A, Maher TM. Interstitial lung diseases. *The Lancet*. 2022 Sep 3;400(10354):769-86.
- Zhang L, Fan QJ, Dang S, Han D, Zhang M, Yan SG, Xin XK, Yu N. CT Quantitative Analysis in Evaluating Type 2 Diabetes Mellitus Complicated with Interstitial Lung Abnormalities. *Current Medical Imaging*. 2025 Jan;21(1):E15734056343395.

CASE NO 1:

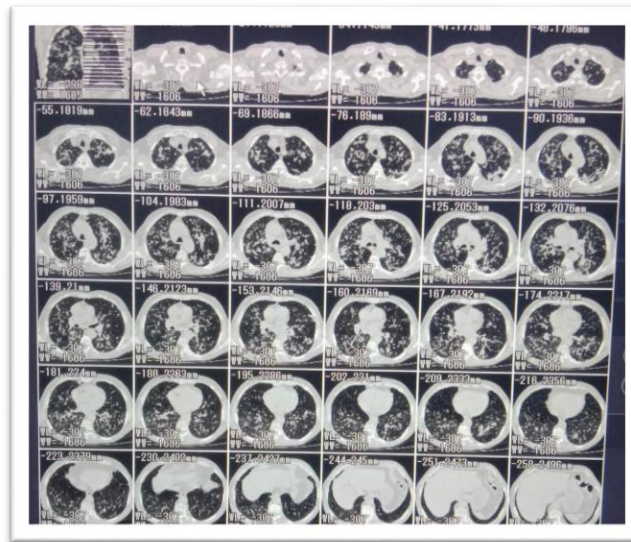


Figure 1

In this case, it shows Diffuse bilateral interstitial and alveolar opacities with ground-glass and consolidative components, predominantly peripheral and basal in distribution
CASE NO: 2

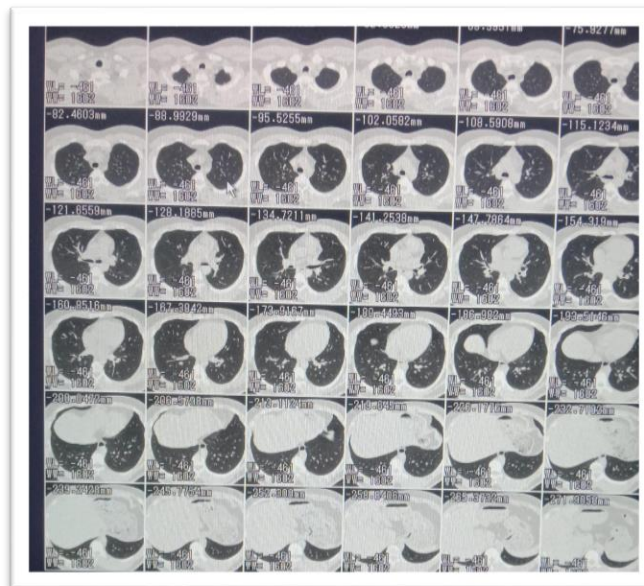
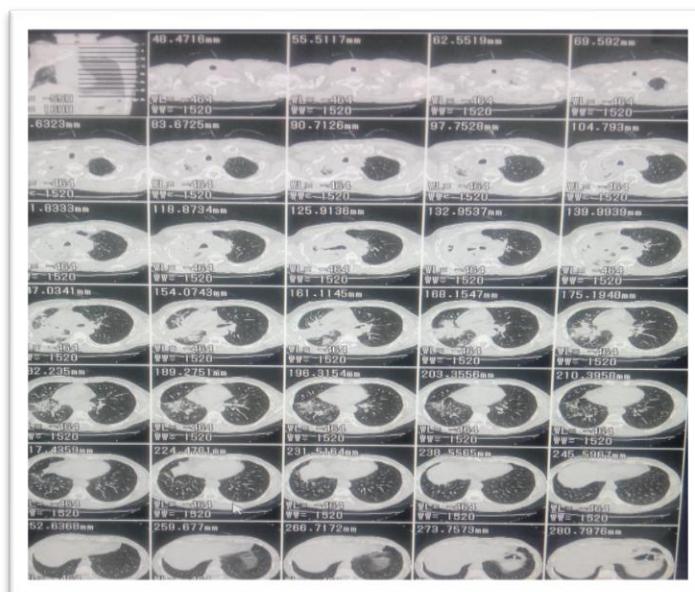


Figure: 2

In this case, it shows mild bilateral peripheral and basal predominant ground-glass and reticular opacities.

**Figure: 3**

In this case, it shows bilateral diffuse patchy interstitial opacities with areas of subpleural involvement,
CASE NO: 4

**Figure: 4**

In this case, it shows bilateral peripheral and basal predominant ground-glass opacities with patchy consolidation.

CASE NO: 5

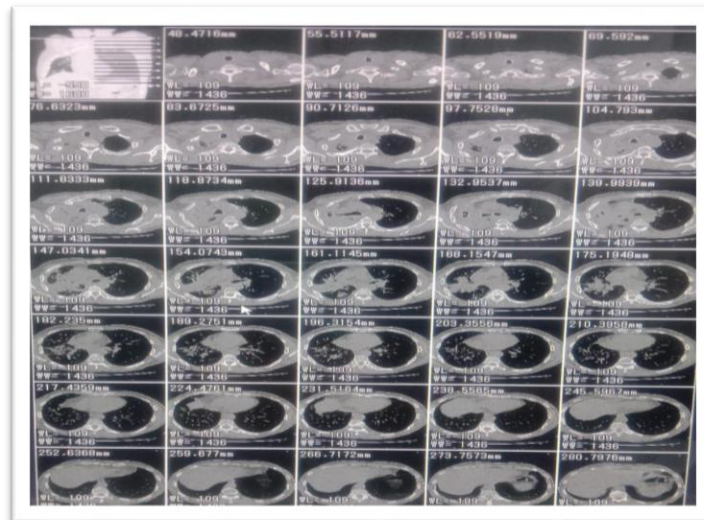


Figure: 5

In this case, it shows bilateral peripheral and basal predominant ground-glass opacities with patchy consolidation.

CASE NO 6

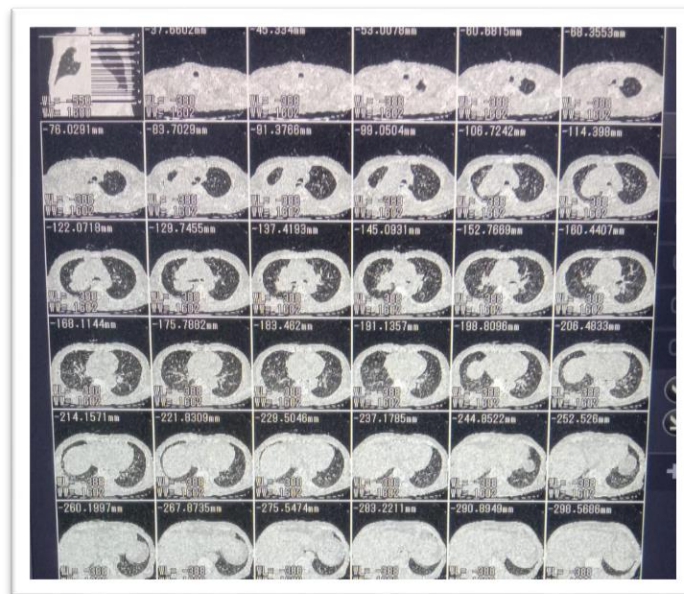


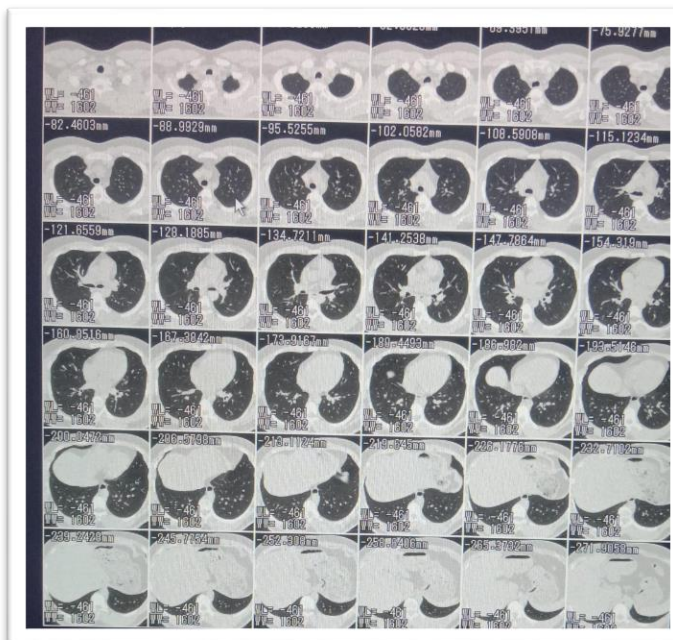
Figure: 6

In this case, it shows bilateral basal and subpleural predominant reticular opacities with ground-glass changes.

**Figure: 7**

In this case, it shows diffuse bilateral ground-glass opacities with possible reticular changes.

CASE NO: 8

**Figure: 8**

In this case, it shows predominantly peripheral and basal reticular opacities with traction bronchiectasis.



Figure: 9

In this case, it shows mild bilateral ground-glass and fine reticular opacities without definite honeycombing.



Figure: 10

In this case, it shows patchy bilateral ground-glass opacities with mild reticulation and subpleural predominance.



Figure: 11

In this case, it shows diffuse bilateral ground-glass opacities with superimposed interlobular septal thickening (crazy paving pattern).



Figure: 12

In this case, it shows bilateral patchy ground-glass opacities with mild reticulation and no definite honeycombing.



Figure: 13

In this case, it shows diffuse bilateral ground-glass opacities with patchy consolidation and minimal fibrosis.



Figure :14

In this case, it shows diffuse bilateral interstitial changes with patchy ground-glass opacities.

