Idiopathic Pulmonary Fibrosis: High Resolution CT Findings Correlated with Pulmonary Function Tests

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Abstract

Objective: Retrospective study was done at Assuit and Al-Azhar Universities, Egypt to demonstrate and compare the high-resolution CT and the pulmonary function tests findings of the idiopathic pulmonary fibrosis.

Patients and Methods: This study was done during 2010-2012 on files of IPF patients. It included: Age, sex, clinical presentation, HRCT findings, and pulmonary function tests. The HRCT scans and the PFT results were retrospectively analysed and compared for 43 patients having IPF.

Results: All patients (43) had a restrictive ventilatory impairment. HRCT showed predominantly reticular abnormalities in five patients. In the remaining 38 cases, HRCT scan showed a mixed pattern (ground-glass and reticular abnormalities and honeycombing). A significant correlation was observed between the overall disease extent in the HRCT and both FVC (r=0.350, p=0.001*) and DLCO (r=0.296, p=0.004*). Significant correlations was also observed with the extent of reticulation and with honeycombing. 18 patients had concurrent emphysema with IPF. The mean diameter of the main pulmonary artery was 30.9mm.

Conclusion: HRCT provides several advantages in the examination of patients with IPF; HRCT is a useful non invasive procedure for confirming the presence of pulmonary hypertension in some patients with diffuse lung disease. Both FVC and DLCO are the pulmonary function studies that could reflect the severity of disease extent and thus disease progression in IPF, as shown by HRCT. Thus, conventional pulmonary function tests have demonstrated value in the assessment of IPF patients.

Key Words: IPF — HRCT — PFTs.

Introduction

IDIOPATHIC pulmonary fibrosis (IPF) is a chronic progressive pulmonary disease of unknown etiology. The diagnosis of IPF is made on the basis of the patient’s history, clinical findings, pulmonary physiology, and imaging results. The diagnosis is one of exclusion of other causes of interstitial lung disease. After nonidiopathic causes are excluded, further investigation of patients with IPF typically reveals radiographic abnormalities and restrictive lung physiology with decreased diffusion capacity. The diagnosis is confirmed with a lung biopsy. Open lung biopsy remains the criterion standard. High resolution computed tomography (HRCT) plays an important role for the diagnosis and management of patients with IPF. Clinical assessment in conjunction with careful review of HRCT scans was 60% sensitive and 97% specific for IPF. However, although HRCT may obviate the need for tissue diagnosis in 60% of patients, surgical lung biopsy is still needed in 40%. Disease progression is slow but continuous. The present therapeutic measures are ineffective and the majority of patients die within 3-8 years because of respiratory failure [1-4].

Since there is still controversy regarding the diagnosis of IPF based on open lung biopsy which could not be done in all suspected cases, this retrospective study was done at Assiut and Al-Azhar Universities, Egypt to demonstrate and compare the high-resolution CTs (HRCT) and the pulmonary function tests (PFT) findings of the IPF.

Patients and Methods

This study reviewed retrospectively files of 43 patients having IPF. The study was done in Assiut University Hospital during 2010-2012. Medical files were prepared and included: Age, sex, clinical presentation, HRCT findings, pulmonary function tests and the severity of pulmonary dysfunction based on the American thoracic society (ATS) criteria [5]. The HRCT scans and the PFT results were analysed and compared.
The population studied consisted of 43 IPF patients (31 men, 12 women), (mean age, 65±1 yr, and range 50-80). 16 were smokers (mean 35±5 pack-years, range 12 to 70), and the remaining 27 had never smoked (12 women, 15 men). The diagnosis of IPF was established without histologic confirmation of the disease. None of the patients had associated collagen vascular disease, occupational, drug induced interstitial lung disease or hypersensitivity pneumonitis. In the studied 43 patients, all patients had dyspnea, 28 had cough, 20 had sputum production, 9 had weight loss, and three had fever. The dyspnea grade ranged from 0 to 4 (mean±SD, 1.9±1.1). Inspiratory crackles were present on physical examination in all patients, finger clubbing in 20, and cyanosis in 21.

**Pulmonary function tests:**

Slow and forced spirometry, single-breath carbon monoxide diffusing capacity of the lung (DLCO), and arterial blood gases at rest while breathing room air were all measured, The forced vital capacity (FVC) and DLCO were expressed as a percentage of the predicted value based on the patients’ height, age, gender and ethnic origin. DLCO values were corrected to hemoglobin [6].

**High-resolution CT scanning:**

**HRCT protocol:** 43 patients underwent helical CT scanning of the chest using a Toshiba X-press scanner. The thin-section CT examinations were performed using 1.5-mm collimation at 15- or 20-mm intervals from the apex of the lung to the diaphragm. The scans were performed with a 1- to 2-s scanning time during breath holding at the end of inspiration. These scans were reconstructed with a high spatial frequency algorithm and viewed at window level (500 to 600 Hounsfield units) and window width (1,400 to 1,600 Hounsfield units) that's appropriate for viewing pulmonary parenchyma [7].

**Intravenous contrast material:** For pulmonary artery diameter measurement, post contrast scans were evaluated in 25 patients who had undergone contrast enhanced CT.100cc non ionic contrast medium was administered manually into the antecubital vein.

**Image evaluation:** The radiologist, without knowledge of any of the clinical, functional and radiographic findings, examined the HRCT scans. The images were assessed for the presence of reticulation (imnumerable, interlinear opacities suggesting a mesh), areas of ground-glass attenuation, (area of hazy increased attenuation without obscuration of the underlying vascular markings) honeycombing (clustered cystic airspaces 3-10mm in diameter with shared well-defined walls) and traction bronchiectasis. Traction bronchiectasis refers to the presence of dilated bronchi within an area of parenchymal opacity (presence of reticulation, ground-glass attenuation, or consolidation). The extent of involvement of the findings was evaluated visually and independently for each level at six predetermined levels: The great vessels, the aortic arch, the tracheal carina, the pulmonary hilae, pulmonary veins and lcm above the right diaphragm [7]. A score was assigned on the basis of the percentage of lung parenchyma that showed evidence of an abnormality and was estimated to the nearest 10% of parenchymal involvement. The overall extent of parenchymal abnormalities was calculated by averaging the scores of the six lung levels to obtain one mean score [8]. The presence of emphysema was determined. Emphysema was defined as areas of decreased attenuation, in comparison with contiguous normal lung and marginalized by a very thin (<1mm) or no wall, and/or multiple bullae (>1cm). The main pulmonary artery diameter (MPAD) was measured in axial cuts and viewed on mediastinal windows. The diameter was measured as the widest diameter perpendicular to the long axis of the artery at the pulmonary artery bifurcation level. The outer limits of the contrast were used to determine the vessel diameter [9].

**Chest radiographs (CXR):** Were examined for manifestations of IPF (bilateral reticular and reticulonodular opacities with small lung volume and typical lower lung zone and peripheral predominance of these infiltrates). No correlation studies were done between CXR and PFTs.

**Statistical Analysis:**

Results are expressed as the mean±SEM. Pearson's correlation test was used to examine the correlation between HRCT and pulmonary function tests. Statistical significance was established at a p-value D105.

**Results**

All patients had a restrictive ventilatory impairment according to forced spirometry. Diffusion capacity of carbon monoxide (DLco) was below the LLR (the lower limit of normal) in all patients. Arterial blood gases at rest were analyzed in all patients. Arterial hypoxemia (PaO2<80mm Hg) was shown in 27 cases and an increase in alveolar-arterial oxygen tension difference (AaP02) (>20mm Hg) was observed in 32 patients.
High-resolution CT Scanning showed predominantly reticular abnormalities in five patients, (Fig. 4). In the remaining 38 cases, HRCT scan showed a mixed pattern with ground-glass, reticular abnormalities and honeycombing, (Fig. 5-7). In 31 of them, the extent of reticular pattern was higher than the extent of ground-glass opacification (GGO), involves upper, middle and lower lobes and not confined to subpleural or basal lung zones. A significant correlation between the overall disease extent in the HRCT and both (FVC) (r=0.350, p=0.001*) and DLCO (r=0.296, p=0.004*) was observed (Fig. 1). In addition, the extent of honeycombing, (Fig. 2) correlated significantly with FVC (r=0.648, p=0.000*)and DLCO (r=0.393, p=0.000*). Also the extent of reticulation was significantly correlated with these two PFT parameters FVC (r=0.373, p=0.000*) and DLCO (r=0.272, p=0.008*), (Fig. 3). On the other hand no significant correlation was found between the extent of GGO and the PFTs, (Table 1).

HRCT scans showed that emphysema was present in 18 cases. Various types of emphysema was observed: Centrilobular, panlobular, Paraseptal and Paracicatricial associated with progressive massive fibrosis (PMF) (Figs. 4,6,7). No differences in FVC or DLCO were observed between the results obtained from patients with or without emphysema.

The main pulmonary artery diameter could be measured on all post contrast CT scans (25 patients) Pulmonary artery hypertension was suspected in 20 patients (Fig. 8). The mean diameter of the main pulmonary artery was 30.9mm.

Chest radiography: In all patients the predominant opacity was small and irregular fine reticular or reticulonodular shadows, 1-3 millimeters, mainly seen at the lung bases. Cardiomegaly and prominent central pulmonary vasculature was also seen in cases of pulmonary artery hypertension (Figs. 5,6B).

Table (1): Correlation between the percentage of extent of HRCT abnormality and the percentage of Pulmonary Function tests in IPF (FEV1, FVC and Diffusion).

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<th>FEV1</th>
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<td></td>
<td>r-value</td>
<td>p-value</td>
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<td>Overall</td>
<td>-0.352</td>
<td>0.001*</td>
<td>-0.350</td>
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<td>Ground glass</td>
<td>-0.014</td>
<td>0.892</td>
<td>0.034</td>
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<td>Honeycombing</td>
<td>-0.549</td>
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<td>Reticular</td>
<td>-0.400</td>
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FEV1: Forced expiratory volume in one second.

Fig. (1): Correlation between overall lung involvement (%) in HRCT and both FVC (%) and DLCO (%) in (IPF)
Fig. (2): Correlation between honey combing (%) in HRCT and both FVC (%) and DLCO (%) in (IPF).

Fig. (3): Correlation between reticular lung involvement (%) in HRCT and both FVC (%) and DLCO (%) in (IPF).

Fig. (4): Axial HRCT in 52 Ys old female patient, shows extensive reticular opacities seen at lcm above the diaphragm level and large right lung para cicatricial emphysematous bulla occupying middle lobe while the chest radiograph shows non specific fine reticular opacities with hyperinflated lungs, low flat diaphragm and sparse vascular pattern in lower lobes.
Fig. (5): Male patient 53 Ys old, CXR reveals reticulonodular opacities with enlarged heart and prominent central pulmonary vasculature (A); HRCT shows mixed pattern of IPF: Patchy ground-glass opacities, fibrosis with reticulation and traction bronchiectasis (white arrows) (B), (C) and dilated pulmonary artery is also seen at both parenchymal and mediastinal windows of the tracheal carina level (C), (D).

Fig. (6 A)

Fig. (6 B)
Fig. (6): 60 Ys old female patient HRCT shows mixed IPF: Extensive reticular opacities, patchy ground glass, honeycombing and paracicatrical emphysema (A). 50 Ys old female patient HRCT at pulmonary veins level showed ground glass, reticulation (mixed pattern), Some lobules appear relatively radiolucent, reflecting air-trapping (white arrows) Cardiomegaly is evident at both HRCT and chest radiograph, (B). 54 Ys old female patient HRCT 1 cm above the diaphragm showed patchy ground glass and reticulation, chest radiograph showed non specific reticulonodular pattern with normal cardiac size (C).

Fig. (7): IPF in 70-Ys-old man. HRCT at oartic arch level (A) shows marked destruction of the right lung, architectural distortion with subsequent shift of the mediastinum to the same side and apical segment of the lower lobe showed collapse or progressive massive fibrosis (mass like fibrosis). 1 cm above the diaphragm (B) bilateral reticulation, traction bronchiectasis (white arrows), and honeycombing (black arrows) and a large pan lobular emphysema occupying the left lower lobe compressing the lingua.
Discussion

Idiopathic pulmonary fibrosis (IPF) is a challenging clinical entity because virtually every aspect of the disease (i.e., pathogenesis, diagnosis, management) remains controversial. Diagnostic uncertainties created in part by the multiple different ways in which physicians have approached IPF (i.e., the availability of appropriate lung biopsy specimens and accurate medical histories) and the variability in the natural history and response to therapy of IPF have contributed to the confusion inherent in this topic. Historically, IPF (or cryptogenic fibrosing alveolitis, as it has been known in Europe) is an inflammatory reaction in the alveolar walls with a mononuclear alveolar exudates and a tendency to progressive fibrosis. In the most advanced stage there is complete destruction of the alveolar architecture. It was originally presumed to be a single clinical entity distinguished by a constellation of symptoms and signs that include middle age patients present with exertional dyspnea and a nonproductive cough. Characteristically they have clubbed fingers, widespread basal crepitations and restrictive pattern on pulmonary function tests. Recently, IPF is defined as a specific form of chronic fibrosing interstitial pneumonia of unknown cause, limited to the lungs and associated with a histologic pattern of usual interstitial pneumonia (UIP). The terms "usual interstitial pneumonitis" and "idiopathic pulmonary fibrosis" are often used interchangeably.

Fig. (8): Axial HRCT, two cases of IPF with pachy ground glass, fine reticulations, air trapping, increased diameters of the central pulmonary vessels and right ventricular enlargement: Case (A) female patient 54 Ys old, MPAD was 30mm. Case (B) male patient 50 Ys old, MPAD was 32mm.
HRCT provides excellent imaging of the lung in the axial plane and is particularly valuable for detecting early infiltrative lung disease. The term GGO is used to describe a generalized haziness of the lungs as if a light veil had been drawn across the film, it represents the earliest detectable radiographic abnormality and at this stage of the disease a response to steroids is most likely. As the disease progresses with the development of fibrosis, HRCT reveals a reticular pattern which characteristically involves the subpleural regions and lung bases and is pachy in distribution. Finally ring shadows represent cystic spaces in the lung and on summation give a honeycomb appearance in advanced disease [12]. The characteristic HRCT manifestations of IPF are reticulation and honeycombing, which are usually bilateral and symmetric, although it can sometimes be asymmetric. The reticular pattern results from a combination of intralobular lines and irregular septal thickening, but the lobular architecture is often so distorted that these may be impossible to recognize. Dilated and distorted bronchioles (i.e, traction bronchiolectasis) and bronchi (traction bronchiectasis) are frequently visible within the areas of reticulation. Honeycomb cysts usually range from 2 to 20mm in diameter. They typically seem to share walls on HRCT and usually manifest as a single row or several layers of cysts in the subpleural lung [13].

GGO tends to be associated with an improved prognosis. In a prospective study of 38 cases of biopsy-proven IPF, Gay et al. [14]. Showed that the extent of GGO on HRCT correlated with greater likelihood of response to treatment and that HRCT was superior to pulmonary function tests and open lung biopsy in predicting response to therapy. Although GGO may reflect the presence of potentially reversible active inflammation, it may also result from interstitial fibrosis and microscopic honeycombing below the resolution of HRCT. GGO should be considered as consistent with active inflammation only when there are no superimposed findings of fibrosis such as reticulation, architectural distortion, or traction bronchiectasis, and this was present in the present study as all cases showed GGO associated with reticulation, traction bronchiectasis as the main HRCT findings indicating that our cases were not early cases. Other potential causes of GGO in patients who have IPF include honeycomb cysts filled with secretions, superimposed diffuse alveolar damage, or a superimposed complication such as an infection or drug reaction [15].

The characteristic basal and peripheral predominance of the abnormalities on HRCT scans is an important clue to the diagnosis of IPF. It is important to realize, however, that the fibrosis tends to involve all lobes. In a study by Hunninghake et al. [16], 85% of patients with IPF had reticulation in the upper lobes [17], and this coincides with our findings where the reticular pattern involves mostly all lobes.

HRCT scanning has dramatically altered the diagnostic approach to patients with diffuse parenchymal lung diseases (DPLDs). The technique allows a detailed evaluation of the lung parenchyma by using narrow section collimation with slice thickness of 1 to 2mm that have been reconstructed with an algorithm that maximizes spatial resolution. One retrospective study [18] reported that abnormalities can be identified when they are not visible on a CXR and that HRCT scans can provide an accurate diagnosis in a large proportion of cases of DPLD while decreasing interreader variability, this was improved when HRCT scan data were complemented by clinical history and interaction with expert radiologists.

Hunninghake et al. [19] reported that in approximately 50% of cases, the characteristic (HRCT) manifestations of IPF are sufficient to allow a confident diagnosis of IPF, obviating lung biopsy in many patients. It is important to realize, however, that in the remaining 50% of patients the HRCT findings are relatively nonspecific and may mimic those of other interstitial lung diseases.

IPF remains the most common of the idiopathic interstitial pneumonias and portends a poor prognosis. Significant strides have been made in the approach to diagnosis and in the ability to predict outcome in the last few years. Furthermore, HRCT scanning may aid in determining prognosis and identifying disease progression. The appropriate use of the HRCT scan requires a multidisciplinary iterative approach incorporating all available data to reach a final diagnosis [20].

The relationship between HRCT findings and pulmonary function tests in IPF has been investigated by several authors. Wells and coworkers [21] analyzed the changes in serial CT scans and showed that improvement in pulmonary function tests was associated with regression of GGO. Other studies [22] have shown that HRCT findings significantly correlate with several functional parameters, such as static lung volumes, FEV1, or DLCO. However, some of these studies [23,24] evaluated only the significance of GGO or reticular pattern, but not the overall extent of the disease in HRCT. The findings of the present study confirm the results
of Staples and coworkers [25] and Wells and coworkers [21] who observed that DLCO was the pulmonary function parameter that best correlated with the overall extent of disease in HRCT. In the present study the results obtained were based on HRCT findings, which represent a sensitive method for determining the extent of parenchymal alterations in IPF. Open lung biopsy was not performed in our cases. Recent guidelines largely acknowledge that in clinical practice the risks of an open lung biopsy may outweigh the benefits of establishing a definite diagnosis of IPF [26]. However the method of scoring the extent of parenchymal HRCT abnormalities limits the present study because it is subjective, semiquantitative and may not have reflected the exact extent of the disease. To measure accurately the overall volume involved would be cumbersome. Simply to add the percentage of involvement in each section and then divide by the number of sections would be incorrect since different sections contain markedly different volumes of lung parenchyma. The method is not ideal but it is the best available. Differences between scanners, effect of gravity, and, particularly, depth of respiration lead to wide variations in the measured values [27,28]. According to our results, conventional functional tests can offer significant information about the extent of parenchymal abnormalities, and thus, indicate that it is probably not necessary to repeat HRCT as a routine test to evaluate the progression of the disease owing to the radiation burden and their high economic cost. Baseline PFTs may provide an estimate of prognosis and serial PFTs provides valuable information in determining disease progression and response to therapy.

It has been shown that the presence of emphysema can influence both FVC and DLCO values in patients with IPF [29]. In this study, however, there were no differences between the results obtained from patients with or without emphysema. Our explanation was that in the presence of both conditions (IPF and emphysema) the pattern will vary according to the predominant pathology, so we found non significant difference between both groups, also spirometric abnormalities in emphysema alone is obstructive, in the present study it was restrictive because the IPF was the predominant pathology as indicated by HRCT. Other studies stated that: With emphysema, the lung volumes and flow rates may be normal due to the counteracting physiological effects of emphysema and fibrosis. HRCT will verify the coexistence of these two processes in the lung [30]. It is still unclear whether the appearance of both processes is simply the coincidence of two diseases with distinct underlying mechanisms but a common risk factor, or whether there is a shared pathway in certain individuals that results in both fibrosis and emphysema after exposure to cigarette smoke [31]. Smoking is the most important risk factor for the development of emphysema, however other factors as air pollution, genetic factor as alphalantitrypsin deficiency, occupations are other risk factors considered for the development of emphysema and this could explain that two cases with emphysema were nonsmokers in the present study.

In combined pulmonary fibrosis and emphysema (CPFE) syndrome, HRCT reveals signs of IPF consisting of honeycombing, reticular opacities predominating on the basis and in the subpleural zones, distortion of the architecture of the lung and/or traction bronchiectasis. Ground glass opacity is more frequent in this syndrome than in IPF. There is almost always centrilobular and paraseptal emphysema. The latter represents a characteristic finding of the CPFE. Bullae are also a common finding on HRCT [32]. All these findings were detected in 18 cases of emphysema associated with IPF in the present study.

Pulmonary hypertension has been shown to be substantially more common and more severe in CPFE than in either IPF or emphysema alone. It is possible that the prevalence and severity of pulmonary hypertension is simply an additive effect of two disease processes independently associated with pulmonary hypertension. A more intriguing possibility is that there is a common process in susceptible individuals, perhaps mediated through chronic inflammation induced by cigarette smoke that results in vascular remodelling in addition to fibrosis and emphysema [31].

Several studies have been performed to measure the pulmonary artery diameter, and have shown that the increase in the main pulmonary artery diameter (MPAD) is a reliable indicator of pulmonary hypertension [22-24]. OCR is a poorly reliable method for the examination of PAD. Several factors contribute to the problem: Superposition of the mediastinal and hilar structures, architectural distortion and magnification differences [9].

The main pulmonary artery diameter was measured in 25 patients who had undergone contrast enhanced CT scans. Pulmonary hypertension was suspected in 20 out of 25 patients (18 patients had CPFE including 16 smokers). The mean diameter of the MPAD was 30.9mm. Kuriyama et al. [33] determined that a main pulmonary artery of 29 mm or larger, as shown on a CT scan, has a sensitivity of 69% and a specificity of 100% for pre-
dicting pulmonary hypertension. In addition, Tan et al. [22] demonstrated that individuals with intrinsic lung disease can be identified as having pulmonary hypertension if the main pulmonary artery is greater than 29mm. However, our measurements were made from the hard copy rather than from the console image and therefore probably overestimated the diameter of the pulmonary artery. Another limitation of the study was that we measure pulmonary artery on axial slices of single slice helical CT, measurements would be more accurate with Multiplanar reformats of multidetector CT which was not available on this retrospective study.

Once pulmonary hypertension has developed, both CPFE and IPF have a similarly poor prognosis and options for medical therapy are unproven [31]. However, early in the disease course, the recognition of emphysema in patients with IPF has critical implications for monitoring. The present study emphasizes the importance of taking into account the presence of concurrent emphysema for the interpretation of pulmonary function tests in IPF. Future studies on concurrent emphysema with IPF are needed.

The chest X-ray is a useful screening test for the diagnosis of IPF/UIP reveals diffuse reticular opacities; however, it lacks diagnostic specificity. In the present study we examined the CXR for manifestations of IPF and we observed that CXR findings could not be measured quantitatively and thus was not correlated with PFTs. This coincides with other studies who stated that: Although the CXR may show a characteristic pattern of changes, it correlates poorly with the clinical and functional deterioration in these patients, and it may be normal in the presence of moderately severe clinical and functional impairment [37]. As previously demonstrated by others, no correlation was found between the extent of disease as assessed by the CXR and the reduction of lung volumes, expiratory flows, and diffusing capacity [35,36]. While chest-X-ray cannot be considered the primary imaging modality to evaluate patients with IPF, it can be used to roughly assess the extension of the disease and to exclude major complications, it allows a panoramic view, being at the same time cost-safe and relatively time-saving [37].

HRCT findings are significantly more sensitive and specific for the diagnosis of idiopathic pulmonary fibrosis. On the HRCT scan, the lack of superimposition of parenchymal structures on the HRCT scan allows clear visualization of areas of fibrosis and distinction from normal parenchyma. Air-density spaces (honeycomb cysts) are also more readily detected on HRCT scans. On the HRCT scan, areas with interstitial disease can be clearly separated from normal parenchyma and the percentage of involved parenchyma easily estimated visually on a single section; however, estimation of the percentage of overall parenchyma involved in a given patient is necessarily subjective.

In summary, contrary to chest radiograph, HRCT provides several advantages in the examination of patients with IPF. CT is a useful non invasive procedure for confirming the presence of pulmonary hypertension in some patients with diffuse lung disease. When the problem of superimposition of densities is reduced, the true distribution of the disease becomes apparent, and since IPF is typically a patchy disease, the HRCT scan theoretically can best direct the surgeon to the site most appropriate for biopsy. The main limitations of HRCT are higher radiation exposure and cost.

We conclude that HRCT not only provides a superior pictorial assessment of the distribution and extent of disease but also correlates better than the chest radiograph does with the clinical and functional severity of IPF. HRCT plays an important role in the initial diagnosis, the assessment of disease extent, and the assessment of some complications.

Due to the presence of correlation between HRCT and both DLco and FVC, the present study also shows that both forced vital capacity and single-breath diffusing capacity for carbon monoxide are the pulmonary function studies that could reflect the severity of disease extent and thus disease progression in IPF, as shown by HRCT. Thus, conventional pulmonary function tests have demonstrated value in the assessment of IPF patients.

**References**


